ORNL
Oak Ridge
National
Laboratory

Operated by
Union Carbide Corporation for the
Department of Energy
Oak Ridge, Tennessee 37830

ORNL/EIS-106

EPA
United States
Environmental Protection
Agency

Office of Research and Development
Health Effects Research Laboratory
Cincinnati, Ohio 45268

EPA-600/1-78-026

12/[illegible]78

REVIEWS OF THE ENVIRONMENTAL EFFECTS OF POLLUTANTS:

IV. Cadmium

DISTRIBUTION OF THIS DOCUMENT IS UNLIMITED

ORNL/EIS-106
EPA-600/1-78-026
June 1978

Contract No. W-7405-eng-26

REVIEWS OF THE ENVIRONMENTAL EFFECTS OF POLLUTANTS: IV. CADMIUM

by

Anna S. Hammons, James Edward Huff, Helen M. Braunstein,
John S. Drury, Carole R. Shriner, Eric B. Lewis,
Bradford L. Whitfield, and Leigh E. Towill

Information Center Complex, Information Division
Oak Ridge National Laboratory
Oak Ridge, Tennessee 37830

operated by
Union Carbide Corporation
for the
Department of Energy

Technical Reviewer
Ernest C. Foulkes
University of Cincinnati
Cincinnati, Ohio

NOTICE
This report was prepared as an account of work sponsored by the United States Government. Neither the United States nor the United States Department of Energy, nor any of their employees, nor any of their contractors, subcontractors, or their employees, makes any warranty, express or implied, or assumes any legal liability or responsibility for the accuracy, completeness or usefulness of any information, apparatus, product or process disclosed, or represents that its use would not infringe privately owned rights.

Interagency Agreement No. D5-0403

Project Officer
Jerry F. Stara
Office of Program Operations
Health Effects Research Laboratory
Cincinnati, Ohio 45268

MASTER

June 1978

Prepared for
HEALTH EFFECTS RESEARCH LABORATORY
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
CINCINNATI, OHIO 45268

Printed in the United States of America. Available from
National Technical Information Service
U.S. Department of Commerce
5285 Port Royal Road, Springfield, Virginia 22161
Price: Printed Copy $11.00; Microfiche $3.00

This report was prepared as an account of work sponsored by an agency of the United States Government. Neither the United States Government nor any agency thereof, nor any of their employees, contractors, subcontractors, or their employees, makes any warranty, express or implied, nor assumes any legal liability or responsibility for any third party's use or the results of such use of any information, apparatus, product or process disclosed in this report, nor represents that its use by such third party would not infringe privately owned rights.

This report has been reviewed by the Health Effects Research Laboratory, U.S. Environmental Protection Agency, and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the U.S. Environmental Protection Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

CONTENTS

Figures v
Tables. ix
Foreword. xvi
Acknowledgments xvii
Abstract. xix
1. Summary 1
  1.1 Discussion of Findings 1
  1.2 Conclusions. 4
2. Physical and Chemical Properties and Analysis 6
  2.1 Summary. 6
  2.2 Physical Characteristics 7
  2.3 Chemical Characteristics 8
    2.3.1 Cadmium in Air. 8
    2.3.2 Cadmium in Water. 10
    2.3.3 Cadmium in Soils. 22
    2.3.4 Cadmium in Biological Systems 26
  2.4 Analysis for Cadmium 27
    2.4.1 Considerations in Analysis. 27
    2.4.2 Analytical Procedures 28
    2.4.3 Comparison of Analytical Methods. 38
3. Biological Aspects in Microorganisms. 50
  3.1 Summary. 50
  3.2 Metabolism 50
    3.2.1 Bacteria. 50
    3.2.2 Yeast 51
    3.2.3 Plankton. 51
  3.3 Effects. 51
    3.3.1 Bacteria. 51
    3.3.2 Algae 53
    3.3.3 Protozoa. 54
    3.3.4 Fungi 54
4. Biological Aspects in Plants. 58
  4.1 Summary. 58
  4.2 Metabolic Processes. 58
    4.2.1 Uptake and Absorption 58
    4.2.2 Translocation, Distribution, and Accumulation 67
    4.2.3 Elimination 80
  4.3 Effects. 80
5. Effects of Cadmium on Aquatic Animals and Birds 90
  5.1 Summary. 90
  5.2 Fish and Other Aquatic Organisms 90
    5.2.1 Metabolism. 90
    5.2.2 Effects 95
  5.3 Birds. 101
    5.3.1 Metabolism. 103
    5.3.2 Effects 108
6. Effects on Terrestrial Mammals Including Man. 113
  6.1 Summary. 113
  6.2 Metabolism 114

        6.2.1 Uptake and Absorption 114
        6.2.2 Transport and Distribution. 119
        6.2.3 Excretion 125
        6.2.4 Biological Half-Time. 128
        6.2.5 Normal Levels 129
        6.2.6 Blood, Urine, and Feces as Indicators of Body Burden 134
    6.3 Effects. 134
        6.3.1 Mechanisms of Action. 134
        6.3.2 Toxic Effects 137
7. Environmental Distribution and Transformation 170
    7.1 Summary. 170
    7.2 Trends in Production and Usage 171
    7.3 Distribution of Cadmium in the Environment 175
        7.3.1 Sources of Pollution. 175
        7.3.2 Distribution in Air 179
        7.3.3 Distribution in Soils 189
        7.3.4 Distribution in Water 193
        7.3.5 Distribution in Sediments 199
    7.4 Environmental Fate 200
        7.4.1 Mobility and Persistence in Air 200
        7.4.2 Mobility and Persistence in Soil. 202
        7.4.3 Mobility and Persistence in Water and Sediments 204
    7.5 Waste Management 204
8. Environmental Interactions and Their Consequences 217
    8.1 Summary. 217
    8.2 Environmental Cycling of Cadmium 217
    8.3 Food Chains. 217
        8.3.1 Cadmium in Foods. 217
        8.3.2 Cadmium in Cigarettes 223
        8.3.3 Terrestrial Ecosystems. 223
        8.3.4 Aquatic Ecosystems. 227
9. Assessment of the Effects of Cadmium in the Environment 239
    9.1 Introduction 239
    9.2 Nature, Sources, and Extent of Cadmium Pollution 239
        9.2.1 Analytical Problems 239
        9.2.2 Domestic Production and Import. 240
        9.2.3 Uses of Cadmium 240
        9.2.4 Flow into the Environment 240
    9.3 Cadmium in the Human Food Chain. 243
    9.4 Toxicity and Health Effects of Cadmium 244
    9.5 Standards for Environmental and Occupational Exposures 245
    9.6 Environmental Hazards Posed by Present and Projected Concentrations of Cadmium in the Environment 247
    9.7 Research Needs 248
        9.7.1 Improvement in Identification and Analysis of Low Concentrations of Cadmium 248
        9.7.2 Development of Measures to Minimize and to Adequately Dispose of Wastes. 248
        9.7.3 Further Information Related to Biological Effects . . 249

FIGURES

2.1 The vapor pressures of cadmium and zinc vs temperature. . . . 7

2.2 The solubility of cadmium vs pH at different carbonate concentrations at 20°C. 12

2.3 The distribution of cadmium complexes in solution in the presence of increasing concentrations of various ligands. . 14

2.4 Rates of sorption of cadmium on hydrated oxides of Mn(IV), Fe(III), and Al(III). 18

2.5 Corrosion of cadmium as a function of pH in carbonate-free solutions 19

2.6 Effect of total carbonate species concentration on rate of cadmium corrosion 20

2.7 Effect of external galvanic coupling on rate of cadmium corrosion 21

2.8 Diagrammatic representation of a silicate clay crystal. . . . 23

2.9 Percent ^{109}Cd retained by montmorillonites in 0.01 *M* Ca(NO$_3$)$_2$. 23

2.10 Adsorption of cations by humus colloids 24

2.11 Influence of pH on the cation exchange capacity of montmorillonite and humus 25

2.12 Fitting a two-site mechanism to observed complexing of Cd^{2+} by captina humic acid. 26

2.13 Correlation of absorbency with concentration of cadmium and of silver-titratable mercapto groups. 27

3.1 Effect of cadmium concentration on ^{14}CO$_2$ evolution by *Escherichia coli* (10^8 cells). 53

3.2 Growth rate of *Selenastrum capricornutum* as a function of cadmium concentration in culture medium 54

4.1 Mean cadmium concentrations in radish tops and roots grown on unlimed (pH 4.1) and limed (pH 5.5) soils containing various concentrations of added cadmium 60

4.2 Cadmium content of plants grown in 200 mg cadmium per 1000 g soil 69

4.3 Uptake of ^{109}Cd by fescue grass and by three species of mosses following application as $CdCl_2$ in simulated rainfall 72

4.4 Uptake of ^{109}Cd by fescue grass and by three species of mosses following application as CdO in simulated rainfall 73

5.1 Toxicity of cadmium in hard water to rainbow trout 98

5.2 Uptake of ^{109}Cd by chipping sparrows (feeding on tagged bird seed). 103

5.3 Biological turnover of ^{109}Cd by chipping sparrows following 21 days of feeding on tagged bird seed 104

6.1 Fraction of particles deposited in the three respiratory tract compartments as a function of particle diameter. . . . 115

6.2 Concentrations of cadmium in plasma and blood cells in mice given a single subcutaneous injection of $^{109}CdCl_2$ and killed various times after injection 120

6.3 Whole-body retention of ^{115m}Cd in rats following a single exposure by different routes of administration 129

6.4 Cadmium content in renal cortex as a function of age, exposure, and geographical location. 131

6.5 Cadmium content in liver as a function of age, exposure, and geographical location. 132

6.6 Cadmium content in renal cortex as a function of age and geographical location. 133

6.7 Effect of oral cadmium administration on milk production of lactating holstein cows. 138

7.1 Schematic flowsheet for recovery of cadmium as a by-product of zinc and lead recovery. 172

7.2 Societal flow of cadmium in the United States, 1968. 173

7.3 Rates, routes, and reservoirs of cadmium in the environment. . 179

7.4 Location of U.S. deposits of lead, lead-zinc, and zinc-lead ore 180

7.5 Location and approximate outputs (1968) of major U.S. zinc production mines 180

7.6 Surface geochemical cycle of cadmium 201

8.1 Cadmium concentrations of surface waters, soils, and foods and estimated dose levels resulting in various symptoms and effects in humans. 218

8.2 Cadmium content in various food samples collected in October 1968 and June 1969 in Annaka City, Japan, compared with unpolluted samples. 222

8.3 Generalized food web for man showing cadmium introduction and movement 223

8.4 Cadmium concentration in cigarettes of different ages. 224

8.5 Metal cadmium concentrations (ppm dry weight) in selected trophic levels of a deciduous forest ecosystem in East Tennessee. 227

8.6 Mean activity in stream ecosystem components after labeling with $^{109}CdCl_2$. 234

TABLES

2.1 Some properties of cadmium and its important compounds 9

2.2 Solubility products of various compounds 11

2.3 Stepwise formation constants of various cadmium complexes. . . 12

2.4 Effect of chloride concentration on solubility of cadmium precipitates 16

2.5 Percent recovery of metal salts from platinum crucibles after heating at various temperatures for one hour 28

2.6 Comparison of atomic absorption spectroscopy results on New York City particulate sample on filter paper prepared by wet digestion and dry ashing. 30

2.7 Commonly used instrumental methods for the analysis of cadmium. 33

2.8 Interlaboratory study on trace level analysis of cadmium by atomic absorption spectrometry 40

2.9 Composition of water metals sample used in interlaboratory comparison 41

2.10 Summary of interlaboratory data on manganese, silver, and cadmium analyses of water samples. 42

2.11 Comparison of cadmium concentrations in coal, fly ash, fuel oil, and gasoline by analytical method and by laboratory . . 43

3.1 Cadmium concentrations causing toxicity to various microorganisms 52

4.1 Estimates of cadmium concentrations in some plant tissues. . . 59

4.2 Concentrations of ^{109}Cd in Japanese millet derived from two chemical forms of cadmium at three levels of calcium carbonate. 61

4.3 Correlation coefficients between levels of cadmium and levels of other elements in the same plant part 62

4.4 Cadmium content of pasture species from adjacent fertilized and unfertilized pastures. 64

4.5 Influence of cadmium contamination of soil on cadmium content of various parts of eight crops. 65

BLANK PAGE

4.6 Distribution of cadmium in cereal plants grown in pot culture. 66

4.7 Cadmium content of and uptake by vegetable species grown in pot culture. 68

4.8 Extractable cadmium and zinc contents of roadside soil and vegetation as a function of distance from traffic and depth in the profile 71

4.9 Cadmium content of woody plants growing in New Haven, Connecticut. 74

4.10 Concentrations of cadmium in components of a spruce forest in central Sweden polluted by a local industrial source 75

4.11 Distribution of cadmium in the components of aboveground biomass in *Calluna* and *Erica* ecosystems. 76

4.12 Mean cadmium concentrations in tree, shrub, and grass samples from six vegetation-type areas in Missouri 78

4.13 Mean cadmium concentrations and ash yields in tree and shrub samples from mineralized areas in Colorado 79

4.14 Phytotoxic effects exerted by cadmium on seedling plants of economic importance in Illinois. 81

4.15 Cadmium concentrations in nutrient solutions producing 50% growth reduction in some crops 81

4.16 Effect of cadmium contamination of soil on yield of various parts of eight crops 82

5.1 Cadmium uptake by marine organisms 91

5.2 Distribution of cadmium in organs of rainbow trout reared in cadmium solution (0.0048 ppm). 92

5.3 Cadmium concentrations in bluegill tissues after an 11-month chronic exposure 93

5.4 Average cadmium concentrations in fish from upper Clark Fork River in western Montana 94

5.5 Cadmium levels in organisms from the Illinois River. 95

5.6 Acute doses of cadmium from some aquatic organisms 96

5.7 Cadmium toxicity to some aquatic organisms 97

5.8 Chronic toxic effects of cadmium to aquatic organisms. 99

5.9 Cadmium toxicity to fiddler crabs as a function of salinity and temperature 100

5.10 Comparison of the acute toxicity of a copper, cadmium, and zinc mixture with that of individual metals to fathead minnows 101

5.11 Spawning and hatching success of fathead minnows at various concentrations of copper, cadmium, and zinc 102

5.12 Percentages of whole-body cadmium in chickens at sacrifice. . 105

5.13 Cadmium residues in starlings in the United States, 1971. . . 106

5.14 Concentrations of cadmium in livers of eight adult puffins from the British Isles. 108

6.1 Cadmium levels in organs of Finnish males and females at autopsy 122

6.2 Tissue distribution of ^{109}Cd 14 days after oral or intravenous administration to goats. 124

6.3 Cadmium excretion in urine of normal, unexposed individuals . 125

6.4 Cadmium in hair from boys living in urban areas 127

6.5 Cadmium concentration in whole blood from "normal" persons and exposed workers 130

6.6 Cadmium and zinc values in human liver and kidney 136

6.7 Concentrations of cadmium in kidney cortex of workers exposed to cadmium oxide dust in relation to morphological kidney changes seen at autopsy or biopsy 142

6.8 Zinc and cadmium concentrations in water and snow 145

6.9 Changes in glutamic oxaloacetic transaminase activity following intravenous injection of cadmium into rabbits . . 147

6.10 Cadmium levels in liver and kidney with respect to lung and other cancers 149

6.11 Cadmium levels in liver and kidney with respect to lung cancer and/or emphysema 150

6.12 Cadmium teratogenicity in hamsters. 151

6.13 Symptoms of itai-itai disease 153

7.1 Natural abundance of zinc and cadmium. 171

7.2 Projected U.S. demand for cadmium and zinc in 2000 based on use in 1968 174

7.3 Approximate material balance for cadmium in the United States in 1968. 176

7.4 Cadmium released from various sources in the United States . . 178

7.5 Cadmium concentrations in U.S. urban air, 1969 182

7.6 Nonurban sites showing less than 0.001 μg cadmium per cubic meter of air in 1969 188

7.7 Cadmium in sedimentary rocks 190

7.8 Cadmium in igneous rocks 190

7.9 Cadmium in soils 191

7.10 Cadmium in waters. 194

7.11 Cadmium concentrations and flows in selected rivers. 195

7.12 Regional summary of cadmium in U.S. surface waters 196

7.13 Locations at which U.S. Public Health Service mandatory upper limit for cadmium in drinking water was exceeded . . . 197

7.14 Cadmium cycling budget in Walker Branch watershed, Oak Ridge, Tennessee, 1974. 198

7.15 Range of metal contents in digested sewage sludge. 206

7.16 Average concentrations of metals in raw sludge 206

7.17 Concentration of cadmium in several industrial sludges 207

8.1 Cadmium content and zinc to cadmium ratio in some foods. . . . 220

8.2 Concentrations of cadmium added to field plots as $^{109}Cd(NO_3)_2$ in four grassland community food chain components 225

8.3 Observed retention of cadmium in sediment, shells, and organisms of a synthetic marine microcosm. 230

8.4 Residues of total cadmium in fish from New York State waters, 1969 231

8.5 Cadmium in organisms from waters of the Southampton and The Solent areas of England 233

8.6 Distribution of 284 μCi $^{115m}CdCl_2$ in microcosms 27 days after tagging 234

FOREWORD

A vast amount of published material is accumulating as numerous research investigations are conducted to develop a data base on the adverse effects of environmental pollution. As this information is amassed, it becomes continually more critical to focus on pertinent, well-designed studies. Research data must be summarized and interpreted in order to adequately evaluate the potential hazards of these substances to ecosystems and ultimately to public health. The Reviews of the Environmental Effects of Pollutants (REEPs) series represents an extensive compilation of relevant research and forms an up-to-date compendium of the environmental effect data on selected pollutants.

Reviews of the Environmental Effects of Pollutants: IV. Cadmium includes information on chemical and physical properties; pertinent analytical techniques; transport processes to the environment and subsequent distribution and deposition; impact on microorganisms, plants, and wildlife; toxicologic data in experimental animals including metabolism, toxicity, mutagenicity, teratogenicity, and carcinogenicity; and an assessment of its health effects in man. The large volume of factual information presented in this document is summarized and interpreted in the final chapter, "Environmental Assessment," which presents an overall evaluation of the potential hazard resulting from present concentrations of cadmium in the environment.

The REEPs are intended to serve various technical and administrative personnel within the Agency in the decision-making processes, i.e., in the development of criteria documents and environmental standards, and for other regulatory actions. The breadth of these documents makes them a useful resource for public health personnel, environmental specialists, and control officers. Upon request these documents will be made available to any interested individuals or firms, both in and out of the government. Depending on the supply, the document can be obtained directly by writing to:

Dr. Jerry F. Stara
U.S. Environmental Protection Agency
Health Effects Research Laboratory
26 W. St. Clair Street
Cincinnati, Ohio 45268

R. J. Garner
Director
Health Effects Research Laboratory

ACKNOWLEDGMENTS

The authors are especially grateful to William Fulkerson, Energy Division, Oak Ridge National Laboratory, for reviewing preliminary drafts of this report and offering valuable comments and to Gerald U. Ulrikson, Manager, Information Center Complex, and Jerry F. Stara, EPA Project Officer, for providing consistent guidance and support. The efforts of J. Michael Fielden and Lilabeth S. Cockrum, who collected the bulk of the literature used in this review, are gratefully acknowledged. Gratitude is also expressed to Carol A. Brumley and Anita B. Gill, editors, and Patricia B. Hartman and Donna M. Stokes, typists, for preparing the manuscript for publication. The authors also appreciate the cooperation of the Toxic Materials Information Center, the Toxicology Information Response Center, and the Environmental Mutagen Information Center of the Information Center Complex.

Appreciation is expressed to Bonita M. Smith, Karen L. Blackburn, and Donna J. Sivulka for EPA in-house reviews and editing and for coordinating contractual arrangements. The efforts of Allan Susten and Rosa Raskin in coordinating early processing of the reviews were important in laying the groundwork for document preparation. The advice of Walter E. Grube was valuable in preparation of manuscript drafts. The support of R. John Garner, Director of Health Effects Research Laboratory, is much appreciated. Thanks are also expressed to Carol A. Haynes and Peggy J. Bowman for typing correspondence and corrected reviews.

ABSTRACT

This report is a comprehensive, multidisciplinary review of the health and environmental effects of cadmium and specific cadmium derivatives. More than 500 references are cited.

Background levels of cadmium are typically low. The highest concentrations are most likely found in localized regions of smelters or in industrialized urban areas. Cadmium is introduced into the environment mainly during extraction, refining, and production of metallic cadmium, zinc, lead, and copper; through the wastes generated during other metallurgical processes such as electrolytic plating; through the reprocessing of scrap metal such as cadmium-plated steel; following disposal by combustion; or as solid wastes from consumer items such as paints, nickel-cadmium batteries, and plastics. Other sources of cadmium in the environment are cadmium-containing fungicides, phosphate fertilizers, and municipal sewage sludges.

The cadmium body burden in animals and humans results mainly from the diet. In the United States, the normal intake of cadmium for adult humans is estimated at about 50 μg per day. Tobacco smoke is a significant additional source of cadmium exposure. The kidneys and liver together contain about 50% of the total cadmium body burden.

Acute cadmium poisoning is primarily an occupational problem, generally from inhalation of cadmium fumes or dusts. In the general population, incidents of acute poisoning by inhaled or ingested cadmium or its compounds are relatively rare.

The kidney is the primary target organ for toxicity from prolonged low-level exposure to cadmium. No causal relationship has been established between cadmium exposure and human cancer, although a possible link between cadmium and prostate cancer has been indicated. Cadmium has been shown to be teratogenic in rats, hamsters, and mice, but no such effects have been proven in humans. Cadmium has been reported to increase the frequency of chromosomal aberrations in cultured Chinese hamster ovary cells and in human peripheral leucocytes. More recent studies on individuals exposed to cadmium showed no statistically significant difference in the frequency of chromosomal aberrations from those individuals not exposed to cadmium. A suggested role of cadmium in cardiovascular disease has not been proven.

The major concern about environmental cadmium is the potential effects on the general population. There is no substantial evidence of hazard from current levels of cadmium in air, water, or food. However, because cadmium is a cumulative poison and because present intake provides a relatively small safety margin, there are adequate reasons for concern over possible future increases in background levels.

This report was submitted in partial fulfillment of Interagency Agreement No. D5-0403 between the Department of Energy and the U.S. Environmental Protection Agency. The draft report was submitted for review in September 1975. The final report was completed in July 1977.

SECTION 1

SUMMARY

1.1 DISCUSSION OF FINDINGS

Cadmium is a soft, bluish-silver-white metal which is harder than tin but softer than zinc; it melts at 321°C and boils at 765°C (Section 2.2). Cadmium is relatively rare, occurring most commonly as an impurity in the commercially important zinc ore sphalerite (zinc sulfide) (Section 7.2). Major uses for cadmium are in electroplating, pigment production, plastics stabilizers, alloys, and nickel-cadmium batteries. Minor uses include electrical contact production, curing of rubber, use in fungicides, and use in solid-state systems (Section 7.2).

Present background levels of cadmium are typically low. For example, uncontaminated soils contain 0.4 ppm cadmium or less, and many open rural waters contain less than 1.0 ppb (Section 7.3). Cadmium content of air in rural or uncontaminated urban areas is usually below detection levels. The highest concentrations of cadmium are generally found in localized regions of smelters or in industrialized urban areas. Distribution and concentrations vary with season and distance from the source. In the vicinity of smelters, soil contains about 10 to 100 ppm; waters near industrial areas contain about 1 to 100 ppb. Most of the cadmium in natural waters is contained in sediments and suspended particles. Cadmium in urban air varies from below detectable limits to 0.150 $\mu g/m^3$ with means usually near 0.005 to 0.015 $\mu g/m^3$.

Background levels are exceeded because of mining, refining, and processing procedures and as a result of municipal waste disposal. Major sources of industrial liquid and solid wastes containing cadmium are electroplating (liquid slurry and sludge), nickel-cadmium battery manufacturing (sludge from baths), paint manufacturing (sludge), and paint in old containers (residues to municipal dumps). Cadmium dusts and fumes from metal extraction, refining, and processing industries constitute a major pollution source of atmospheric cadmium. Lesser sources of cadmium pollution are combustion of fossil fuels, phosphate fertilizers with a high cadmium content, wearing of tires, and combustion of lubrication oil. Threshold limit values for cadmium exposure in industrial air in the United States as established by the American Conference of Governmental Industrial Hygienists are 50 $\mu g/m^3$ for cadmium fume and 200 $\mu g/m^3$ for cadmium dust.

A variety of analytical methods sensitive to as low as 1 ppb are available for determining cadmium in environmental media (Section 2.4.2): spectrophotometric colorimetry, emission spectroscopy, atomic absorption spectrophotometry, neutron activation, polarography and anodic stripping voltammetry, spark-source mass spectrometry, and isotope dilution mass spectrometry. At present, however, no one method satisfies the requirements for accuracy, reliability, and sensitivity while at the same time being simple, rapid, inexpensive, and requiring small samples. Thus, no single analytical method can be characterized as superior for the analysis of cadmium in every circumstance.

Zooplankton and phytoplankton apparently can remove and concentrate trace metals from water by direct absorption. Cadmium is toxic at different concentrations to several species of bacteria, yeast, and fungi. Generally, cadmium levels causing toxic effects in bacteria (0.2 to 6 ppm) are several times higher than concentrations normally detected in industrially polluted waters (1 to 100 ppb) (Sections 3.1 and 7.3.4).

Cadmium accumulation in plants at present background levels is usually less than 1 ppm. Little is known about cadmium concentrations needed to reduce plant yields; however, plants growing in contaminated soils contain abnormally high cadmium levels which may be detrimental to growth. Cadmium contamination of soils can result from industrial sources such as smelters or from the use of phosphate fertilizers and sewage sludge. Increased uptake of cadmium by plants directly increases dietary cadmium levels in animals and humans.

Cadmium may be transported to all regions of the plant, but it most often accumulates in the roots. Cadmium content in plants usually reflects environmental cadmium levels. Several factors may stimulate uptake of cadmium by plants: increased soil acidity, increased soil temperature, the presence of small amounts of zinc, or oxidation of insoluble cadmium sulfide by soil microorganisms (Section 4.2.1). In controlled experiments, cadmium produces five major effects on plants: altered ion absorption and release, altered stomatal function, reduced chlorophyll content, reduced cell turgor, and reduced photosynthesis.

Cadmium levels found in animal tissues under normal conditions are usually not high enough to be considered toxic. The primary concern arising from environmental exposure of animals and man centers on effects from low-level chronic exposure. Little information is available on cadmium levels in wild or domestic animals. Evidence suggesting that cadmium concentrates in food chains is fragmentary. Cadmium accumulation in shellfish generally is higher than cadmium accumulation in fish (Section 5.2.1.1).

In the nonoccupational environment, oral intake represents the primary source of the cadmium accumulated in the body; most food and water supplies contain some cadmium (Section 6.2.1.2). The average daily cadmium intake for persons on American diets can be reasonably estimated at about 50 μg from all sources. In the United States, cigarette smokers and persons occupationally exposed to atmospheric cadmium have higher cadmium body burdens than nonsmokers or persons not occupationally exposed (Sections 6.2.1.1 and 8.3.2). Generally, little cadmium has been detected in newborns. Levels increase with age and vary among individuals, depending on diet, environment, and smoking habits (Section 6.2.5.2). Although cadmium has been found in kidneys and other organs in populations all over the world, it does not appear to be a micronutrient.

Cadmium is absorbed through both the respiratory and gastrointestinal tracts. Nutritional factors such as protein or vitamin D deficiencies may increase cadmium absorption. Cadmium is transported from the site of absorption via the bloodstream; most of the cadmium in the bloodstream of exposed workers can be found in the red blood cells (Section 6.2.2.1).

Chronically exposed workers maintain the highest blood cadmium levels; these concentrations have been shown to slowly decrease in workers after exposure stops. Half-times for cadmium in blood approximate six months.

One-third of the total cadmium body burden is in the kidneys (Section 6.2.2.2). Liver and kidneys together contain about 50% of the total body burden, which generally lies between 15 and 30 mg for a 70-kg person. Biological half-times for cadmium based on body burden and excretion data for groups from various countries are estimated at 13 to 47 years (Section 6.2.4). Cadmium is excreted primarily through urine and feces.

The protein metallothionein (thionein plus a bound metal) contains many sulfhydryl groups; it is synthesized in response to metal exposure and binds a major portion of cadmium in the tissues (Section 6.3.1.1). The precise function of metallothionein, if any, in cadmium metabolism remains uncertain. Cadmium also reacts with many other ligands, including several important enzyme systems. The nature of the ligands determines the toxicity of the metal; thus, the nephrotoxicity of cadmium is increased by low-molecular-weight sulfhydryl compounds. Some toxic effects of the metal arise from its competition with zinc at the level of zinc-requiring enzymes; certain acute effects of cadmium can indeed be prevented or reduced by excess zinc. Selenium is also an effective cadmium antagonist.

Acute cadmium poisoning via inhalation continues to be an occupational rather than an environmental hazard (Section 6.3.2.2.1). Inhaled freshly generated cadmium fume is the most toxic form. Acute cadmium poisoning due to ingestion is rare (Section 6.3.2.2.2); use of cadmium in food or drink containers is prohibited.

Signs and symptoms of acute cadmium poisoning generally begin one-half to one hour after ingestion and include: (1) severe nausea, vomiting, diarrhea, abdominal cramps, and salivation; (2) headache, muscular cramps, vertigo, and (rarely) convulsions; (3) exhaustion, collapse, shock, and death; or (4) gradual appearance of kidney and liver damage resulting in death in one to two weeks from acute renal failure.

The critical organ with respect to prolonged, low-level exposure to cadmium is the kidney. Itai-itai disease, believed by many investigators to result from chronic cadmium exposure due to industrial contamination of the environment, reflects primarily renal tubular dysfunction plus osteomalacia and osteoporosis (Section 6.3.2.4.7).

Proteinuria and kidney stones are commonly found in cadmium workers. Other renal changes may include glucosuria, amino aciduria, and decreased urine concentrating ability. No critical level of cadmium in the renal cortex has been generally accepted, but at approximately 200 ppm wet weight kidney damage may be observed. Moderate, apparently reversible anemia has been associated with occupational exposure to cadmium oxide dusts and fumes (Section 6.3.2.3.2.2).

Hypertension can be induced experimentally in certain animal species by administration of cadmium; some epidemiological data indicate a positive correlation between hypertension and cadmium in humans; however, a relationship between the two has not been proven.

Experimental animal data suggest that cadmium may cause testicular damage in humans; however, no direct evidence exists (Section 6.3.2.4.2). Limited data indicate a possible correlation between cadmium exposure and cancer of the prostate (Section 6.3.2.4.4). Although no teratogenic effects of cadmium have been reported in humans, cadmium has been shown to be teratogenic in rats, mice, and hamsters. Cadmium-induced mutagenesis has not been experimentally demonstrated; however, an increased number of chromosomal abnormalities were found in peripheral leukocytes from itai-itai patients and in cultured human leukocytes exposed to cadmium sulfide.

The major concern surrounding environmental cadmium stems from potential effects on the general population from chronic, low-level exposure. Emphasis is placed on the effects of soil contamination on plant uptake of cadmium and the resultant effect on animals and humans through diet.

1.2 CONCLUSIONS

1. No one method is uniquely superior for the analysis of cadmium in various materials.

2. Background levels of cadmium are generally low; higher concentrations occur near smelters or in industrialized urban areas. The metal may be present at relatively high concentrations in a variety of industrial and municipal wastewaters.

3. Cadmium is a relatively volatile metal, and significant amounts are contributed to the atmosphere from smelting operations and the combustion of fossil fuels.

4. Cadmium may move among air, biomass, soil, and water; much of the cadmium in water finds its way into sediments.

5. Although cadmium moves within the ecosystem, significant biomagnification along the food chain seems limited; some mollusks are an exception.

6. In sufficient concentration, cadmium is toxic to all forms of life, including microorganisms, higher plants and animals, and man.

7. Information is lacking on the concentrations of cadmium necessary to reduce plant yields, but in general the cadmium levels in plants reflect that in soil. Actual uptake by a given species depends on pH and other chemical characteristics of the soil.

8. Cadmium uptake by plants, especially mosses, may also reflect the concentration of the element in air.

9. Application of cadmium-containing fertilizers and sewage sludges to agricultural lands may lead to significant increases in the cadmium content of the crops.

10. Cadmium in the body of animals and man under normal conditions is derived primarily from the diet; most food and water supplies contain at least traces of the element.

11. An additional contribution to the body burden of cadmium in the general population is made by cigarette smoking.

12. In the occupational setting, significant pulmonary uptake of cadmium dusts or fumes may further elevate the body burden.

13. The biological half-life of cadmium is very long so that its concentration in the body increases with age; the metal therefore acts as a cumulative poison.

14. The primary target organ of cadmium is the kidney; a major portion of renal (and hepatic) cadmium is present in the form of metallothionein, a low-molecular-weight metalloprotein of undefined function.

15. Cadmium readily reacts with sulfhydryl groups and may compete, especially with zinc, for binding sites on proteins. In this manner, the metal may inhibit a variety of enzymatic reactions.

16. A primary role of cadmium is suspected in some human diseases, including itai-itai disease; whether the metal plays a part in the etiology of human hypertension, carcinogenesis, mutagenesis, or teratogenesis remains uncertain.

SECTION 2

PHYSICAL AND CHEMICAL PROPERTIES AND ANALYSIS

2.1 SUMMARY

Cadmium possesses the size, charge, and Group II behavior of calcium and the complexing properties of zinc. Adsorption reactions, complex formation with different ligands, the volatility of the element, and the water solubility of its salts are important factors controlling distribution in the environment. In natural waters, for instance, the presence of carbonate, hydroxide, or sulfide ions may be instrumental in removing cadmium from solution, while ammonia, chloride, or cyanide may maintain the solubility of cadmium. In soils, cadmium mobility and accessibility appear to depend on adsorption by humic material and clays coated with hydrous oxide. Adsorption by humic matter is strongly pH dependent. All fractions of the soil communicate with one another and with natural waters, providing the pathway for movement of free cadmium ions. Organocadmium compounds are unstable in air and water, preventing their presence in the natural environment.

The exact chemistry of cadmium in vivo is still uncertain. The metal is bound to a variety of proteins, especially the low-molecular-weight thioneins in liver and kidney.

A variety of analytical techniques are available for the determination of cadmium in environmental matrices down to the 1-ppb level. Although this level of sensitivity is adequate for most samples, it frequently can be achieved only at the expense of costly, time-consuming preconcentration steps. There is a continuing need for more rapid and inexpensive methods which do not require pretreatment of the samples. Flameless atomic absorption spectrometry, anodic stripping voltammetry, and differential pulse polarography represent excellent advances in this direction, but procedural standardization of these relatively new techniques is required.

Errors associated with the analysis of cadmium at concentration levels near 1 ppb may result as much from sampling techniques, sample storage, and handling procedures as from instrumental variance. Information relative to these factors is not currently available for sample matrices commonly encountered in environmental work. Standard methods similar to those published for water and wastewater should be explored and widely adopted.

Essentially all published determinations of cadmium in environmental samples refer to total cadmium; in general, no attempt is made to identify particular cadmium species which may exist in the sample. Although the chemistry of cadmium is relatively simple, numerous complexes exist, and some may have relevance to the impact of cadmium on the environment.

2.2 PHYSICAL CHARACTERISTICS

Cadmium is a soft, bluish-silver-white metal, harder than tin but softer than zinc; it can be cut with a knife (Chizhikov, 1966). Cadmium has low melting and boiling points (321°C and 765°C respectively); the vapor pressure of the liquid metal between these temperatures is shown in Figure 2.1. Although imperceptible at ambient temperature, the vapor pressure of metallic cadmium rises rapidly with increased temperature; an orange-yellow vapor is emitted. This vapor may be discharged during metal refining or during combustion of fossil fuels or other materials and represents a major source of atmospheric cadmium (Goeller, Hise, and Flora, 1973; Hise and Fulkerson, 1973). Upon contact with air, cadmium vapor is oxidized to CdO (Section 2.3.1).

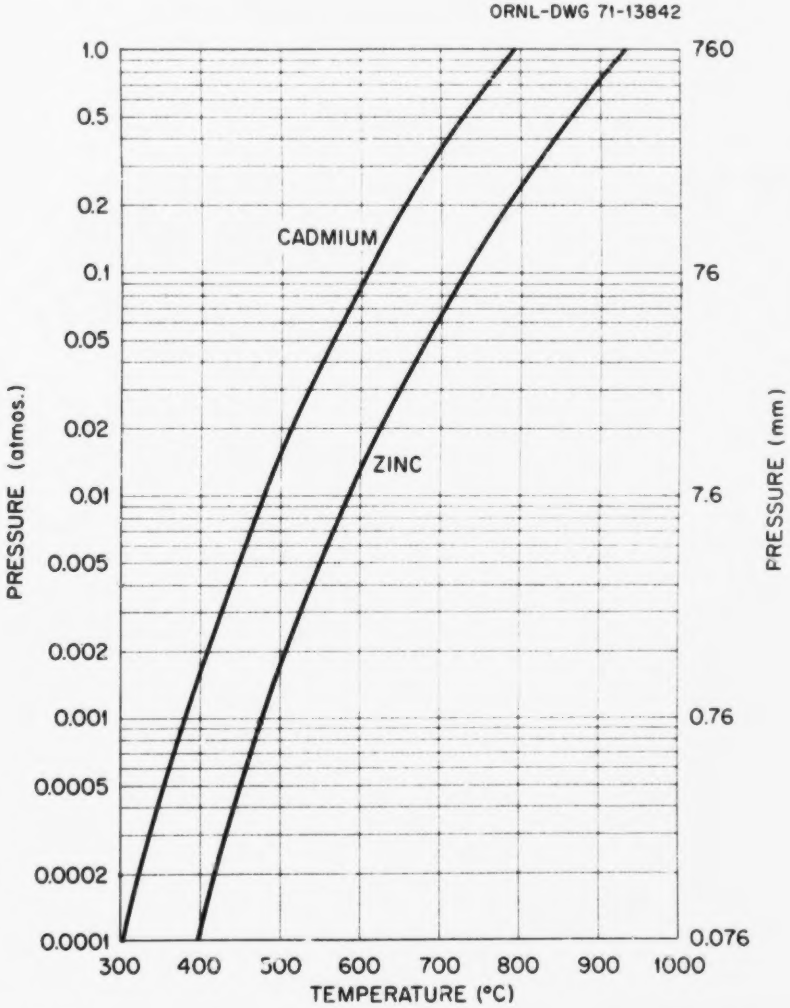


Figure 2.1. The vapor pressures of cadmium and zinc vs temperature. Source: Baes, 1973, Figure III-3, p. 35.

Some properties of cadmium and of its important compounds are shown in Table 2.1. The solubility of many of these compounds is noteworthy in connection with the chemical behavior of cadmium in the aqueous environment (Section 2.3.2).

2.3 CHEMICAL CHARACTERISTICS

The chemistry of cadmium is inseparable from that of zinc. Cadmium is universally associated with zinc in natural deposits; both possess the same outer-electron configuration $[nd^{10}(n + 1)s^2]$ and participate qualitatively, if not quantitatively, in the same hydrolysis and complexation reactions. An unknown amount of cadmium occurs as an impurity in zinc and escapes to the environment whenever zinc escapes. In fact, estimations of cadmium concentrations (in the absence of direct data) are often possible from the fairly constant cadmium-to-zinc abundance ratio (Hem, 1972).

The corrosion behavior of cadmium and zinc in air and water are important in dictating some of the uses of these metals and, thereby, their prevalence in the environment. Cadmium corrodes slightly in air but forms a protective surface film that prevents further corrosion; zinc behaves similarly. This low corrosion renders both zinc (with its customary cadmium impurity) and cadmium industrially valuable as protective surface platings for corrosion-susceptible steel.

Zinc and cadmium react readily with acids to produce hydrogen:

$$Zn + 2H^+ = Zn^{2+} + H_2 \ ,$$

$$Cd + 2H^+ = Cd^{2+} + H_2 \ ,$$

but the analogous reaction with pure water at ambient temperatures is exceedingly slow (Baes, 1973). Thus, until the introduction of copper and plastic water pipe, zinc (containing cadmium) was used widely to galvanize iron water pipe. Some of this piping is still functional, posing a possible contamination problem, particularly in soft-water areas (Section 2.3.2.4 and Table 6.8).

2.3.1 Cadmium in Air

Although the exact forms of cadmium in air are still uncertain, presumably cadmium oxide constitutes the largest portion of airborne cadmium (Friberg et al., 1974). Cadmium vapor, emitted during man-moderated thermal processes, is converted to the oxide on contact with air,

$$Cd + \tfrac{1}{2}O_2 = CdO \ .$$

CdO is also directly released into the air during a variety of combustion processes such as cigarette smoking (Menden et al., 1972).

Likewise, cadmium sulfate, which does not occur naturally, is a conversion product found in atmospheric emissions from thermal processes involving materials containing cadmium and sulfur (International Agency for Research

TABLE 2.1. SOME PROPERTIES OF CADMIUM AND ITS IMPORTANT COMPOUNDS

Compound	Primary use or occurrence	Formula	Molecular weight (g/mole)	Density (g/ml)	Physical state, 20°C	Melting point (°C)	Boiling point (°C)	Solubility in water, 20°C (g/liter)	Solubility in other solvents	Acute lethal dose[a]
Cadmium metal	Cadmium nickel batteries	Cd	112.4	8.6	Silver metal	321	765	Insoluble	Soluble in acid and NH_4NO_3	5 mg/m³ is the approximate lethal concentration in man, inhaled as fume
Cadmium oxide	Smelting plant or coal combustion emission	CdO	128.4	7.0	Brown powder	Decomposes at 900		0.00015[b]	Soluble in acid and NH_4 salts	50 mg/m³ is the approximate lethal concentration in man, inhaled; 72 mg/kg, rat, LD_{50} (oral)
Cadmium sulfide	Pigment for plastics and enamels; phosphors	CdS	144.5	4.8	Yellow crystal	1750	Decomposes	0.0013	Soluble in acid, very slightly soluble in NH_4OH	
Cadmium sulfate	Fruit tree fungicide	$CdSO_4$	208.5	4.7	White crystalline	1000		755	Insoluble in acid and alcohol	27 mg/kg, dog, LD_{50} (subcutaneous)
Cadmium carbonate	Turf treatment	$CdCO_3$	172.4	4.3	White powder or crystalline	Decomposes below 500		0.001	Soluble in acid and KCN, NH_4 salts	
Cadmium chloride	Turf grass fungicide	$CdCl_2$	183.3	4.0	Colorless crystal	568	960	1400	15.2 g/liter in alcohol	88 mg/kg, rat, LD_{50} (oral)
Cadmium potassium cyanide	Electroplating	$K_2Cd(CN)_4$	294.7	1.85	Colorless, glassy crystal			333	Insoluble in alcohol	
Cadmium cyanide	Electroplating	$Cd(CN)_2$	164.4		Colorless crystal	Decomposes at 200		17, soluble in hot water	Soluble in acid and KCN	

Sources:
[a] Fulkerson and Goeller, 1973.
[b] Hahne and Kroontje, 1973
Other data compiled from Weast, 1971.

on Cancer, 1973). The sulfide is readily prepared in the laboratory by heating a mixture of cadmium (or cadmium oxide) and sulfur,

$$Cd + S = CdS \ ,$$

whereas the sulfate is the primary stable product of the oxidation of cadmium sulfide at elevated temperatures (Chizhikov, 1966),

$$CdS + 2O_2 = CdSO_4 \ .$$

Cadmium chloride can be generated and released into the atmosphere by the incineration of polyvinyl chloride plastic to which organic cadmium stabilizers are added (International Agency for Research on Cancer, 1973). Cadmium can appear in air as dust, fumes, and mist as a by-product of the refining of zinc, copper, or lead (McCaull, 1971). Dusts of cadmium sulfide have been reported in refinery working atmospheres (International Agency for Research on Cancer, 1973). Cadmium is present in the atmosphere mostly in the form of particulate matter composed of cadmium oxide and cadmium oxide—iron oxide (from zinc refining) with some cadmium sulfide, cadmium sulfate, and cadmium chloride.

2.3.2 Cadmium in Water

The concentration of cadmium in natural waters may be determined by the solubility of cadmium compounds, by the formation of cadmium complexes, and by the removal of cadmium through adsorption.

2.3.2.1 Solubility — Although the solubility of cadmium compounds has been postulated to be the single most important natural process governing cadmium distribution and availability in the aqueous environment (Posselt and Weber, 1971), Harris, Helz, and Cory (1975), Bailey, Helz, and Harris (1975), and Hem (1972) concluded that it is unlikely that precipitation of inorganic compounds controls the cadmium concentration in natural waters. The solubility product constant (K_{sp}), a molar measure of solubility, is the product of the solute ion concentrations in a saturated solution of the substance. For example, for cadmium carbonate ($CdCO_3$),

$$K_{sp} = [Cd^{2+}][(CO_3)^{2-}] \ ,$$

where the square brackets refer to molar concentrations of the ions.

The solubility products of some environmentally important cadmium compounds are listed in Table 2.2 along with those of additional Group II metals. Posselt and Weber (1971) stated that hydroxide and carbonate ions at alkaline pH may limit the amount of cadmium in solution because of the low solubilities of the corresponding cadmium compounds. In regard to water treatment applications, Posselt (1971) suggested that the effect of carbonate on the solubility of cadmium is dramatic. Thus, cadmium removal via carbonate precipitation appears promising, provided that attainment of equilibrium proceeds at a practical rate. Posselt (1971) concluded that slow attainment of equilibrium is probably the most significant factor in many applications of thermodynamic solubility relationships, particularly

TABLE 2.2. SOLUBILITY PRODUCTS OF VARIOUS COMPOUNDS

Metal	Cation, M^{2+}	Hydroxides, $M(OH)_2$ $\log([M^{2+}][OH^-]^2)$	Carbonates, MCO_3 $\log([M^{2+}][CO_3^{2-}])$	Sulfides, MS $\log([M^{2+}][S^{2-}])$
Magnesium	Mg^{2+}	-10.9	-5.4	
Calcium	Ca^{2+}	-5.43	-8.35	
Zinc	Zn^{2+}	-17.0	-10.20	-24.5
Cadmium	Cd^{2+}	-14.4	-12.01	-28.9
Mercury	Hg^{2+}	(-25.5)		-52.7

Source: Adapted from Baes, 1973, Table III-2, p. 37.

in precipitation reactions. Posselt's conclusions emphasizing the importance of solubility in the distribution of cadmium compounds in the aqueous environment were based on laboratory studies conducted under conditions approaching saturation. After completion of field studies, Hem (1972) concluded that it appears unlikely that many river waters approach saturation in respect to cadmium carbonate. Hem (1972) also suggested that biological factors and/or adsorption by stream sediments may affect the levels of heavy metals in natural waters.

2.3.2.1.1 Hydroxide and carbonate — Knowledge of the behavior of cadmium in hard or soft water is important in evaluating the risk of cadmium ingestion from drinking water (Section 6.3.2.3.2). Cadmium carbonate dissolution or deposition depends not only on the thermodynamic relationship between solid and dissolved $CdCO_3$ and its ions, but also on solubility equilibria involving hydroxide ions. For instance, at low metal ion concentrations, cadmium forms at least four soluble hydroxide species (Posselt and Weber, 1971): $CdOH^+$, $Cd(OH)_2$, $Cd(OH)_3^-$, and $Cd(OH)_4^{2-}$, each of which is described by a formation (equilibrium) constant defined by stepwise equilibria as outlined in Section 2.3.2.2 and enumerated in Table 2.3. Use of the measured solubility product and equilibrium constants (as described by Posselt and Weber, 1971) allows the calculation of dissolved cadmium concentrations in a solution containing both carbonate and hydroxide (Figure 2.2). The derived cadmium concentrations are applicable only in the absence of sulfide or complex-forming ions such as chloride. Sulfide ions remove cadmium by formation of insoluble cadmium sulfide (Section 2.3.2.1.3), whereas complex-forming ions maintain cadmium in solution (Section 2.3.2.2). The solid curves in Figure 2.2 correspond to the total CO_2 concentration (the sum of dissolved carbon dioxide, bicarbonate, and carbonate) in moles per liter. The uppermost solid curve corresponds to zero carbonate, indicating control by the hydroxide equilibrium:

$$Cd^{2+} + 2OH^- = Cd(OH)_2 \text{ (solid)} .$$

Precipitation of $Cd(OH)_2$ reportedly occurs at a pH greater than 7.5 and at

TABLE 2.3. STEPWISE FORMATION CONSTANTS OF VARIOUS CADMIUM COMPLEXES

Ligand, L	$\frac{[CdL]}{[Cd^{2+}][L]}$	$\frac{[CdL_2]}{[CdL][L]}$	$\frac{[CdL_3]}{[CdL_2][L]}$	$\frac{[CdL_4]}{[CdL_3][L]}$
Cyanide, CN^-	300,000	140,000	36,000	4,000
Pyrophosphate, $P_2O_7^{4-}$	10,000	200		
Hydroxide, OH^-	8,500	(1,000)	(180)	22
Ammonia, NH_3	550	150	30	10
Sulfate, SO_4^{2-}	130			
Iodide, I^-	120	5.9	140	30
Nitrite, NO_2^-	63	16	6	
Bromide, Br^-	57	4	10	2
Chloride, Cl^-	35	5	1	
Fluoride, F^-	3			

Source: Adapted from Baes, 1973, Table III-4, p. 41.

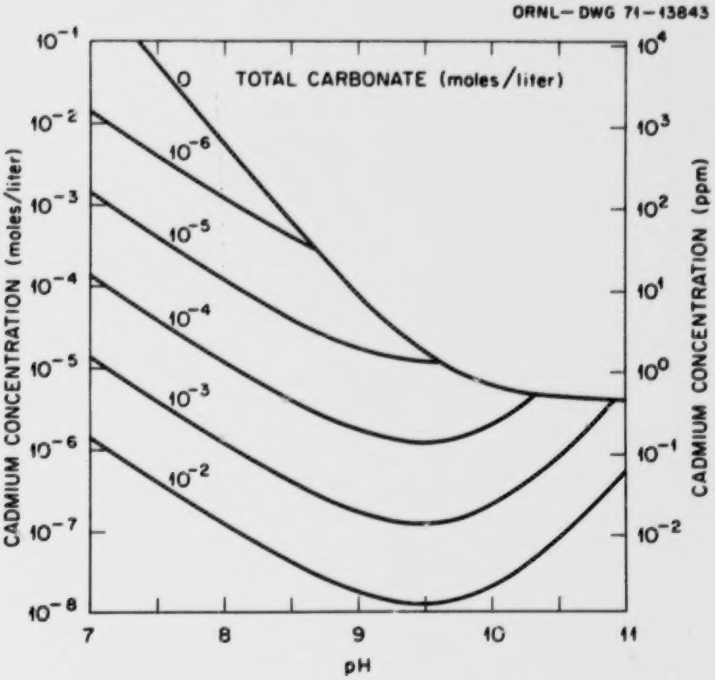


Figure 2.2. The solubility of cadmium vs pH at different carbonate concentrations at 20°C. Source: Baes, 1973, Figure III-6, p. 47.

a hydroxide-to-cadmium ion ratio greater than or equal to 0.05 (Posselt and Weber, 1971).

This precipitation may be suppressed by the presence of relatively large concentrations of complex-forming anions such as iodide, bromide, or chloride (Posselt and Weber, 1971). In fact, even hydroxide becomes a complexing ligand at pH values greater than 9.5 (Figure 2.3) so that, in the absence of carbonate, the cadmium concentration cannot be reduced below 0.5 ppm (Figure 2.2). This greatly exceeds (about 50 times) the U.S. Public Health Service maximum of 0.01 ppm for drinking water (Posselt and Weber, 1971). Reduction to the 0.01-ppm cadmium level, indicated by the dashed line in Figure 2.2, is attained only at dissolved carbonate concentrations greater than 10^{-3} mole/liter. Since the carbonate content of hard water rarely exceeds 4×10^{-3} mole/liter (U.S. Environmental Protection Agency, 1974), water hardness alone does not appear to provide a dependable means for reduction of cadmium concentrations in water to desirable levels. Additionally, only equilibrium thermodynamic considerations have been applied here, and these may easily be overruled by kinetic factors that can hinder deposition of cadmium as the carbonate. Posselt and Weber (1971), in fact, found the final rate of attainment of equilibrium in the precipitation of soluble cadmium from alkaline carbonate solutions rather slow and highly irreproducible.

Hem (1972) compared observed cadmium concentrations in 80 samples of river water with cadmium solubilities calculated from graphs similar to Figure 2.2. The conclusion was drawn that not many river waters are likely to approach saturation with respect to cadmium carbonate. Further evidence that inorganic precipitation does not control the cadmium concentration was provided by Bailey, Helz, and Harris (1975) and Harris, Helz, and Cory (1975).

2.3.2.1.2 Carbonate solid solution — Baes (1973) discussed the likelihood of solid-solution formation, Cd^{2+} replacing Ca^{2+} in precipitated $CaCO_3$ and other calcium-bearing minerals, as a result of similarity in size and charge of the two ions. Although Baes considered the exchange in terms of removal of cadmium from natural waters, solid solutions of Cd^{2+} in $CaCO_3$ may be even more important as a possible reservoir for dissolution of cadmium (Bondietti et al., 1974), considering the very low concentration to be maintained as a drinking water standard.

2.3.2.1.3 Sulfide — If sulfide is present in solution, cadmium should precipitate as solid cadmium sulfide according to the solubility product constant in Table 2.2: $K_{sp} = 1.2 \times 10^{-29}$. In the event of simultaneous discharge of zinc and cadmium into sulfide-containing waters, the Zn^{2+} to Cd^{2+} ratio in water at equilibrium would be about 25,000, the ratio of the solubilities of the sulfides (Baes, 1973). Because of the biological antagonism between zinc and cadmium, this high Zn^{2+} to Cd^{2+} ratio may be as important as the absolute Cd^{2+} intake in preventing cadmium toxicity (Section 6.3.1.2). In fact, as determined by inspection of the solubility product ratios (Table 2.2), the Zn^{2+} concentration in carbonate-containing waters may also exceed that of Cd^{2+} (by a factor of 65). In soft water, where solubility of cadmium is presumably a function of $[OH^-]$, the Cd^{2+} concentration may be greater than that of Zn^{2+}, even at a high pH.

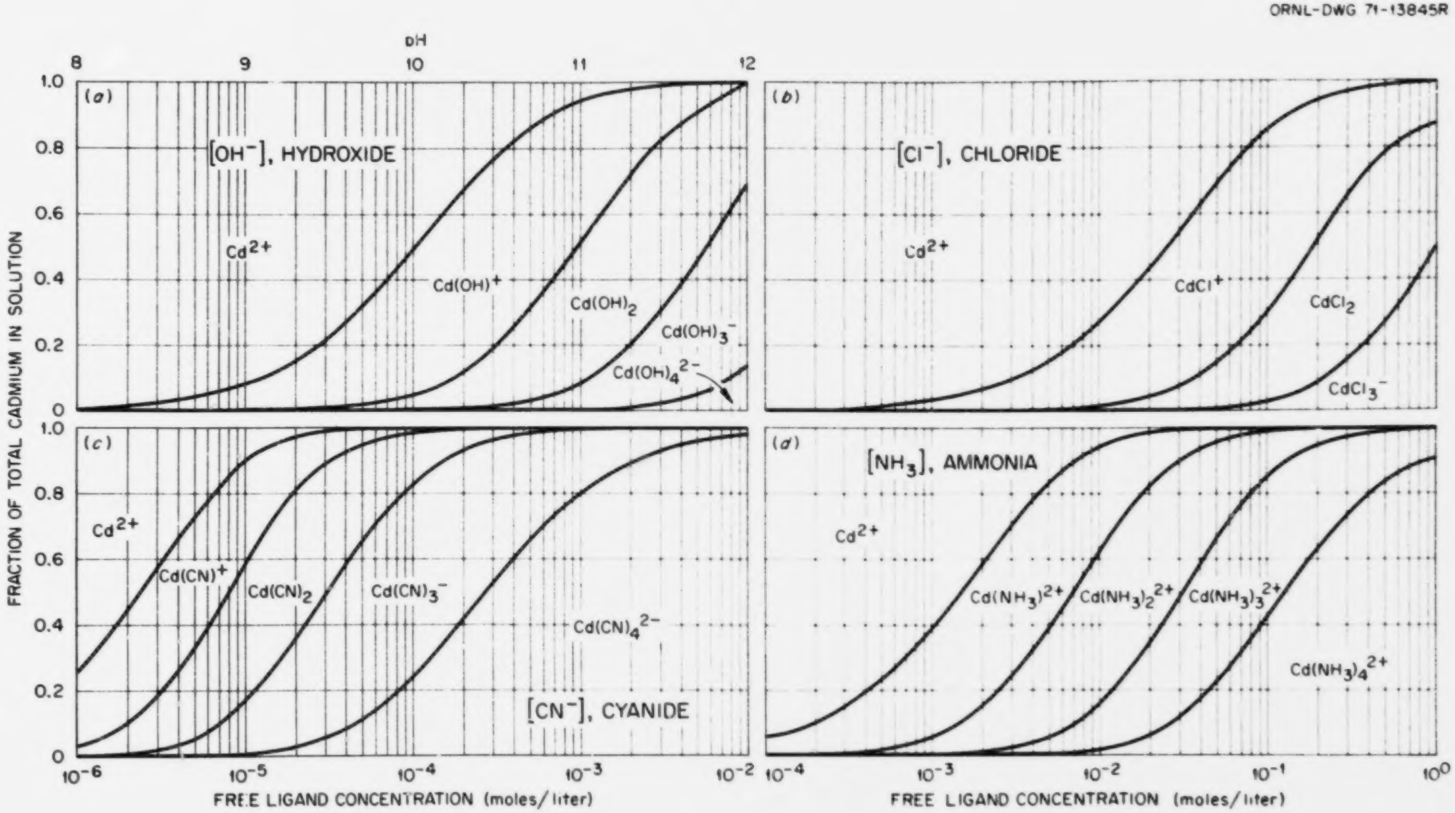


Figure 2.3. The distribution of cadmium complexes in solution in the presence of increasing concentrations of various ligands. Source: Baes, 1973, Figure III-4, p. 42.

According to Hem (1972), precipitation of sulfides may be an important factor in the control of cadmium and zinc in water systems, with the reduced mud in the bottom of some types of lakes and reservoirs constituting a sink for deposition of these metals. Bowen (1966), however, pointed to the activity of aerobic bacteria in oxidizing insoluble sulfides in soils to sulfates, which liberates the cation in a soluble form.

2.3.2.2 Complex Formation — An important property of the Cd^{2+} ion is its ability to complex with negatively charged ions or ligands to form soluble complexes. Complex formation constitutes a serious competitive process which can reduce the extent of precipitation and adsorption, the prime mechanisms for natural removal of cadmium from water systems.

The consecutive addition of ligands (L) is controlled by the stepwise formation constants (K_n) enumerated for a number of ligands in Table 2.3:

$$Cd^{2+} + L^- = CdL^+ , \quad K_1 = \frac{[CdL^+]}{[Cd^{2+}][L^-]} ;$$

$$CdL^+ + L^- = CdL_2 , \quad K_2 = \frac{[CdL_2]}{[CdL^+][L^-]} ;$$

$$CdL_2^+ + L^- = CdL_3 , \quad K_3 = \frac{[CdL_3^-]}{[CdL_2][L^-]} ;$$

$$CdL_3^- + L^- = CdL_4^{2-} , \quad K_4 = \frac{[CdL_4^{2-}]}{[CdL_3^-][L^-]} .$$

Chloride, cyanide, and ammonia form cadmium complexes of environmental importance: chloride because it is prevalent in natural waters and cyanide and ammonia because they appear in industrial wastes and effluents.

2.3.2.2.1 Chloride — Complex formation with chloride becomes a significant factor in solubility considerations at chloride levels greater than 10^{-3} mole/liter (Hahne and Kroontje, 1973). The distribution of chloride complexes for chloride ion concentrations in the range from 1×10^{-4} to 1 mole/liter is shown in the upper right of Figure 2.3. At 0.1 mole/liter chloride, 85% of the cadmium in solution exists as chloride complexes. The effect of this complex formation on the solubility of some cadmium precipitates is given in Table 2.4. Clearly, dramatic increases in cadmium solubility occur as the chloride concentration increases from 0.01 mole/liter to 1 mole/liter. The phosphate salt, which is important when considering cadmium in fertilizer application (Section 7.3.3), shows nearly a 40-fold increase in solubility on contact with a solution of high salinity — a condition encountered in soils as a result, for instance, of runoff from roads from application of chloride salts for snow and ice removal.

TABLE 2.4. EFFECT OF CHLORIDE CONCENTRATION ON SOLUBILITY OF CADMIUM PRECIPITATES

Precipitate	Cadmium values[a]		
	10^{-4} *M* chloride	10^{-2} *M* chloride	1 *M* chloride
$Cd_3(PO_4)_2$	1.001	1.130	39.83
CdS	1.001	1.107	21.55
$Cd(OH)_2$	1.001	1.070	7.745

[a] Values represent the ratio of cadmium dissolved from various salts in the presence of a given chloride concentration to the amount dissolved in absence of chloride.

Source: Adapted from Hahne and Kroontje, 1973, Table 4, p. 450.

2.3.2.2.2 Ammonia — Complex formation of cadmium with ammonia is somewhat stronger than with chloride (Table 2.3 and Figure 2.3). However, because ammonia is rarely present in more than minute amounts — usually at levels much lower than chloride (even in industrial wastes) — its role in forming soluble complexes of cadmium is probably less than that of chloride (Posselt and Weber, 1971). However, since chloride ion and ammonia are independent entities in aqueous solution, their ability to form complexes with cadmium is additive. At pH 9, the calculated solubility of cadmium carbonate is increased fivefold in the simultaneous presence of 0.1 mole/liter chloride ion and 0.005 mole/liter NH_3 (Posselt and Weber, 1971). In the case of ammonia,

$$Cd^{2+} + nNH_3 = Cd(NH_3)_n{}^{2+} \quad (n = 1, 2, 3, \text{ or } 4) ,$$

the divalent cadmium ion is still available to associate with the anion of the insoluble cadmium salt brought into solution by the presence of ammonia.

2.3.2.2.3 Cyanide — Cyanide ion is one of the strongest complexing ligands known. Its appearance in waste streams along with cadmium is a result of electroplating operations, which consume about one-half the cadmium used in the United States (Hem, 1972). Watson (1973) cited a report which stated that cadmium ions are impossible to precipitate as cadmium hydroxide or oxide in the presence of cyanide; pretreatment to destroy this complexing agent is required before cadmium removal in wastewater treatment. This is not surprising since the magnitude of the cyanide association (Table 2.3) exceeds that of chloride by almost 10,000-fold. Fortunately, cyanide can be rapidly and easily broken down (Watson, 1973), allowing removal of cadmium from wastewater following prior destruction of cyanide.

2.3.2.3 Adsorption — Cadmium in water can be adsorbed by clays and muds, by humic and organic materials, and by hydrated oxides of iron and manganese. The phenomenon is of prime importance not only as a mechanism for natural removal of soluble cadmium, but also for chemical treatment of industrial waste streams (Watson, 1973). The discussion of aqueous sorption (adsorption and/or absorption) by clays and humic substances, however, is included in Section 2.3.3 since the sorption behavior in aqueous sediments is similar to that in soils.

Hydrated oxides of manganese and iron are widespread in clays, soils, and sediments, both as coatings on other minerals and as discrete oxide particles (Jenne, 1968). Although little is known about the surface area of the many different naturally formed oxides of iron and manganese, prepared hydrated oxides have large surface areas (200 to 300 m^2/g), a property undoubtedly important in their adsorption ability (Jenne, 1968). Laboratory studies of the rate of sorptive uptake of cadmium by the hydrated oxides of manganese, iron, and aluminum have been reported by Posselt and Weber (1971). Results in Figure 2.4 show that the attainment of sorption equilibrium is very rapid (within 10 min), although rates with aged crystals (for example, in the natural environment) may be much slower (hours, perhaps, rather than minutes). Considerable competition with the sorption process would be expected from ligands with strong complexing capability (for example, cyanide) or from certain organic or humic substances. The sorptive affinity of manganese dioxide for cadmium was superior to that of hydrated iron oxide, but the latter was considered useful at pH values greater than 6. The utility of hydrated aluminum oxide in sorptive removal of cadmium from water was considered doubtful since the sorptive capacity was reported as inferior and the range of applicable pH was limited (Posselt and Weber, 1971). This is not so with aluminum-hydroxide-coated montmorillonite clays, which retain cadmium strongly (Section 2.3.3.1).

2.3.2.4 Corrosion Behavior — Widespread concern has been expressed about the appearance of cadmium in drinking water as a result of corrosion of galvanized iron water pipes (Baes, 1973; Friberg et al., 1974; National Academy of Sciences, 1974; Posselt and Weber, 1971) (Table 6.8 and Section 6.3.2.3.2.3). A more serious situation is likely to exist in cases where galvanized pipes are coupled to copper or brass pipes and fittings; this condition may enhance the rate of corrosion (dissolution of zinc and cadmium) as a result of galvanic effects (Posselt and Weber, 1971). Such galvanic currents between different metals have been implicated in acute cadmium poisoning arising from consumption of soft drinks from a vending machine (Emmelin, 1973); the cadmium was released into the stored water from a high-cadmium alloy used to solder the top and sides of the brass storage tank.

Laboratory studies of the aqueous corrosion chemistry of cadmium (Posselt and Weber, 1974) have identified some cadmium corrosion promoters and inhibitors; a high oxygen content and the coupling of cadmium to copper promote corrosion, whereas a high pH and the presence of carbonate inhibit it. Coupling cadmium directly to less-noble zinc has little effect on the corrosion behavior.

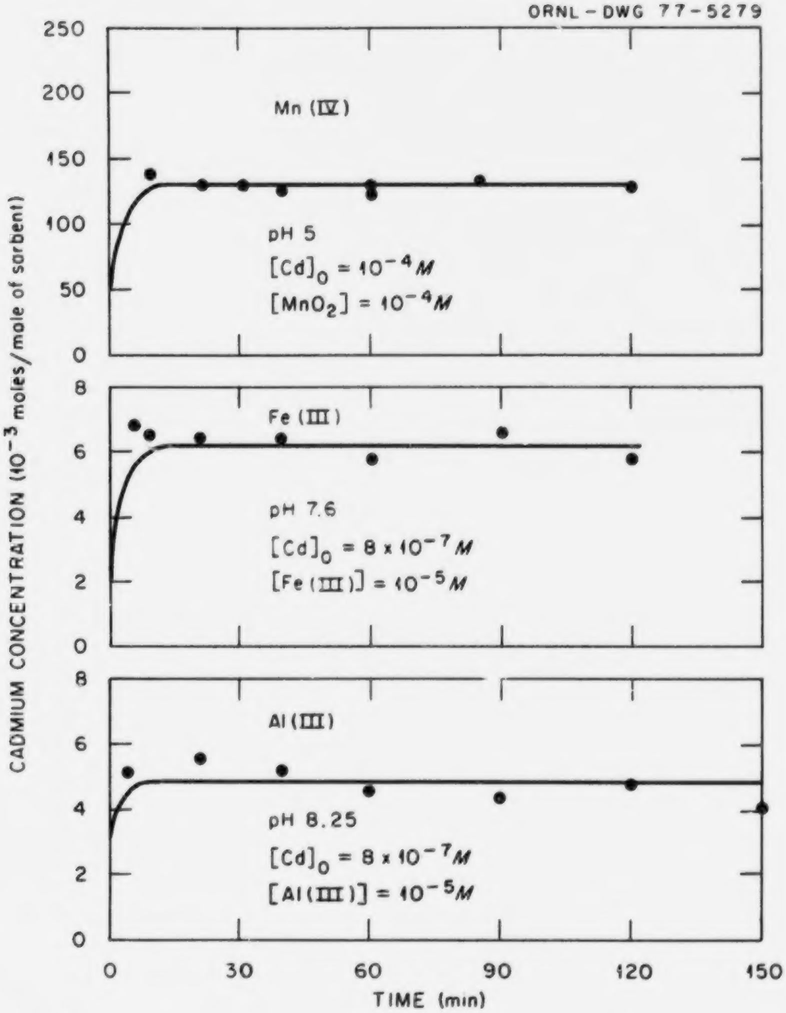


Figure 2.4. Rates of sorption of cadmium on hydrated oxides of Mn(IV), Fe(III), and Al(III). Source: Adapted from Posselt and Weber, 1971, Figure 17, p. 105. Reprinted by permission of the publisher.

The driving force for cadmium dissolution in the presence of oxygen is readily understood in terms of the oxidation potential (E_o) for the two half reactions,

$$Cd = Cd^{2+} + 2e^- \; , \qquad E_o = 0.40 \text{ V} \; ,$$

$$O_2 + 4H^+ + 4e^- = 2H_2O \; , \qquad E_o = 1.23 \text{ V} \; ,$$

and their sum,

$$2Cd + O_2 + 4H^+ = 2Cd^{2+} + 2H_2O \ , \qquad E_o = 1.63\ V.$$

The action is thermodynamically favorable by 1.63 V, but because the reaction requires 2 moles of hydrogen ion for each mole of cadmium oxidized, oxygen attack is decreased sharply by increasing the pH, as shown in Figure 2.5. Cadmium dissolution is reduced by more than an order of magnitude upon increasing the pH from 8.3 to 10.5.

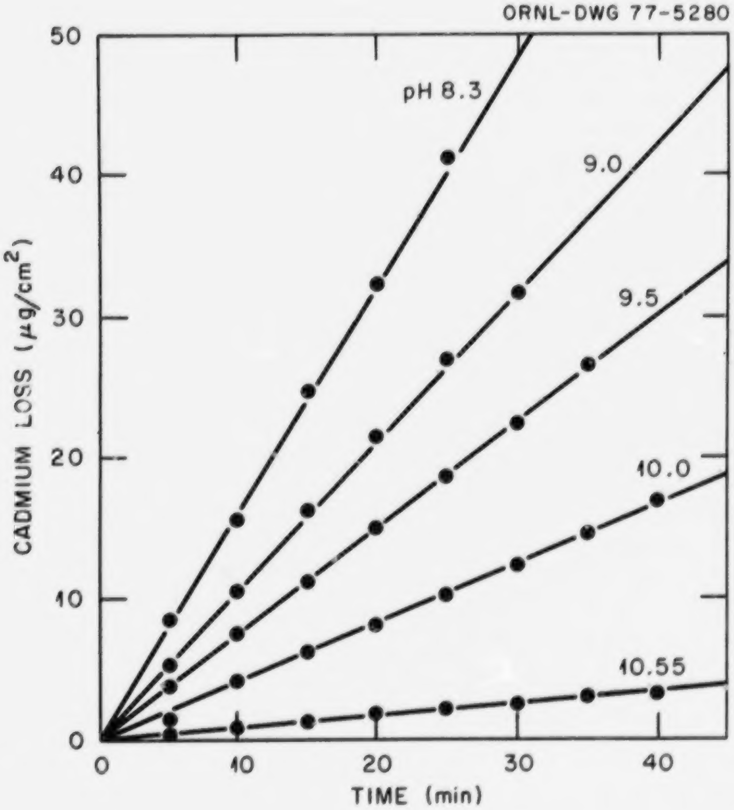


Figure 2.5. Corrosion of cadmium as a function of pH in carbonate-free solutions. Source: Adapted from Posselt and Weber, 1974, Figure 8.2, p. 301. Reprinted by permission of the publisher.

In addition to pH and oxygen concentration, carbonate concentration and copper coupling are important factors in cadmium corrosion; carbonate reduces the corrosion rate. This reduced corrosion rate is thought to occur by gradual formation of a protective film (probably solid $CdCO_3$) that causes a progressive decrease in the cadmium dissolution rate as the film thickens with time. The decrease was interpreted by Posselt and Weber (1974) in terms of an initial and a secondary rate of corrosion; the average primary and secondary rate constants are shown as the upper and lower curves, respectively, of Figure 2.6. The higher the concentration of carbonate, the more effective it is in decreasing the corrosion rate.

ORNL-DWG 77-5281R

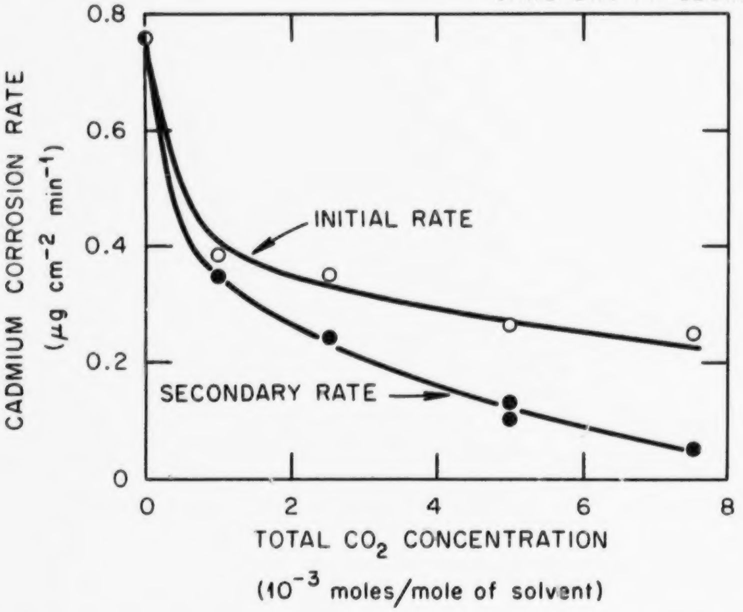


Figure 2.6. Effect of total carbonate species concentration on rate of cadmium corrosion. Source: Adapted from Posselt and Weber, 1974, Figure 8.7, p. 307. Reprinted by permission of the publisher.

Corrosive attack on cadmium is enhanced by external coupling to a nobler metal, such as copper, establishing a galvanic couple:

$$Cd = Cd^{2+} + 2e^- \;, \qquad E_o = 0.403 \text{ V} \;,$$

$$Cu = Cu^{2+} + 2e^- \;, \qquad E_o = 0.337 \text{ V}$$

$$Cd + Cu^{2+} = Cd^{2+} + Cu \;, \qquad E_o = 0.740 \text{ V}$$

Water containing oxygen (or ammonia) in contact with copper metal is likely also to contain traces of dissolved copper (Pourbaix, 1973), which drives the galvanic reaction. This would appear to require an equal corrosion of copper. However, the "leap-frog" mechanism (Pourbaix, 1973) explains the process as occurring by successive deposition and dissolution of copper from one corrosion site to the next:

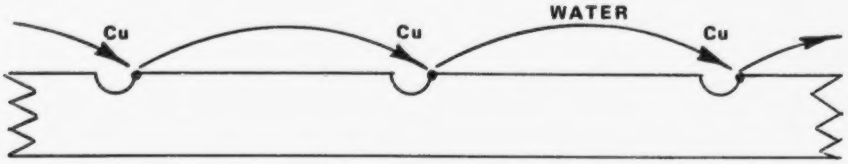


Figure 2.7 shows an almost tenfold increase in the corrosion rate when cadmium is galvanically coupled to copper.

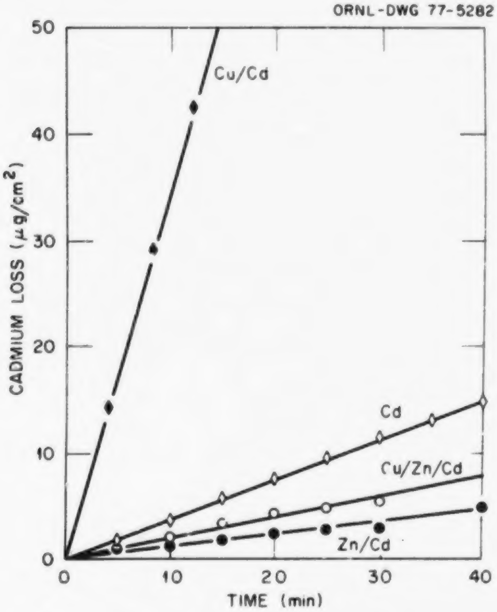


Figure 2.7. Effect of external galvanic coupling on rate of cadmium corrosion. Source: Adapted from Posselt and Weber, 1974, Figure 8.9, p. 311. Reprinted by permission of the publisher.

Coupling to less-noble zinc, which should reverse the galvanic effect, somewhat suppresses corrosion but definitely does not eliminate it (Figure 2.7). This may be due to enhanced passivation of zinc relative to cadmium, which effectively enobles zinc to virtual equality with cadmium (Pourbaix, 1973).

In summary, corrosion of cadmium contained within zinc-galvanized pipe is minimized by hard water and high pH and enhanced by galvanic coupling to copper pipes and fittings. The corrosion proceeds by oxygen reduction, which is hindered at high pH.

2.3.3 Cadmium in Soils

Behavior of cadmium in soils is important in terms of biological availability, including especially uptake by plants. Shacklette (1972) found that the cadmium concentration in normally grown plants is determined by the inherent ability of a plant species to absorb cadmium and by the cadmium concentration in the environment. Studies in solution culture indicated to Van Hook et al. (1973) the importance of the solution chemistry of cadmium in determining cadmium availability to plants. Evaluation of the total amount of cadmium in soils may be important; dissolved cadmium is in dynamic equilibrium with the solid soil and removal of cadmium from the soil solution leads to replenishment from the solid soil due to ionic dissociation (see reviews discussed by Bowen, 1966).

Soils are composed of several fractions: (1) the inorganic mineral portion made up of coarse or colloidal matter; (2) the organic portion, which contains decomposing tissue and humic material; (3) the soil solution or aqueous portion; (4) the air fraction; and (5) the living organisms. Cadmium undoubtedly interacts chemically with all of these but most notably with the colloidal inorganic and humic portions. Solubility interactions and complex formation within the soil solution would be expected to resemble those in other aqueous media (Section 2.3.2). Likewise, interactions with hydrated oxides of manganese, iron, and aluminum — part of the inorganic colloidal fraction of soil — also are expected to be similar to those seen in aqueous solution (Section 2.3.2.3).

2.3.3.1 Clay Minerals — It is important to consider the clay mineral portion of soils in estimating the role of adsorption processes in the availability of heavy metals. Silicate clays are characterized by shape and size of their particles and by their ion exchange properties (Brady, 1974). Unlike sand particles, which are more or less spherical, clay particles are laminated, that is, made up of layers of plates or flakes often no more than a few angstroms thick and 1 to 2 μ across; these dimensions result in a ratio of breadth to thickness of approximately 10,000. Consequently, Brady (1974) conservatively estimated the active surface area due to the clay fraction of an acre-furrow slice of a representative clay loam soil to exceed the land area of Illinois by 40 or 50 times. The sheetlike structure of the clay particles gives rise to an intrinsically produced negative surface charge to which positive cations are attracted (Figure 2.8). Both the external and internal surfaces provide sites for cation adsorption, resulting in a clay colloid particle (micelle) that behaves like a huge anion. The action of the internal surfaces is not only interesting but also is of practical importance. The interplatelet lattice dimension in clay minerals such as vermiculite and montmorillonite is about 1.4 Å (Bowen, 1966). Clays of this type swell when wet, allowing ions such as K^+ (radius, 1.33 Å) and NH_4^+ (radius, 1.48 Å) to permeate between layers. On drying, the layers come together again and are held tightly in place by electrostatic forces; the ions are fixed in place. The retention of cadmium on coated and uncoated montmorillonite is shown in Figure 2.9. Clays coated with hydrated oxides (a common natural occurrence) have increased adsorption and retention of cadmium even under the competitive influence of Ca^{2+}, a behavior not unlike that observed in aqueous solution (Section 2.3.2.3).

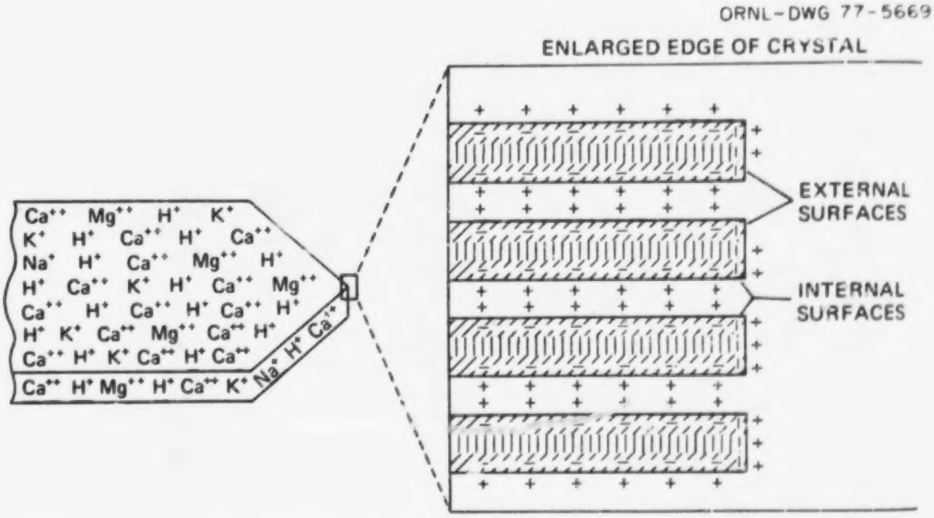

ORNL-DWG 77-5669
ENLARGED EDGE OF CRYSTAL
EXTERNAL SURFACES
INTERNAL SURFACES
Ca++ Mg++ H+ K+
K+ H+ Ca++ H+ Ca++
Na+ H+ Ca++ Mg++ H+
H+ Ca++ K+ H+ Ca++ Mg++
Ca++ H+ Ca++ H+ Ca++ H+
H+ K+ Ca++ Mg++ Ca++ H+
Ca++ H+ K+ Ca++ H+ Ca++
Ca++ H+ Mg++ H+ Ca++ K+ Na+ H+ Ca++

Figure 2.8. Diagrammatic representation of a silicate clay crystal. Source: Brady, 1974, Figure 4.2, p. 74.

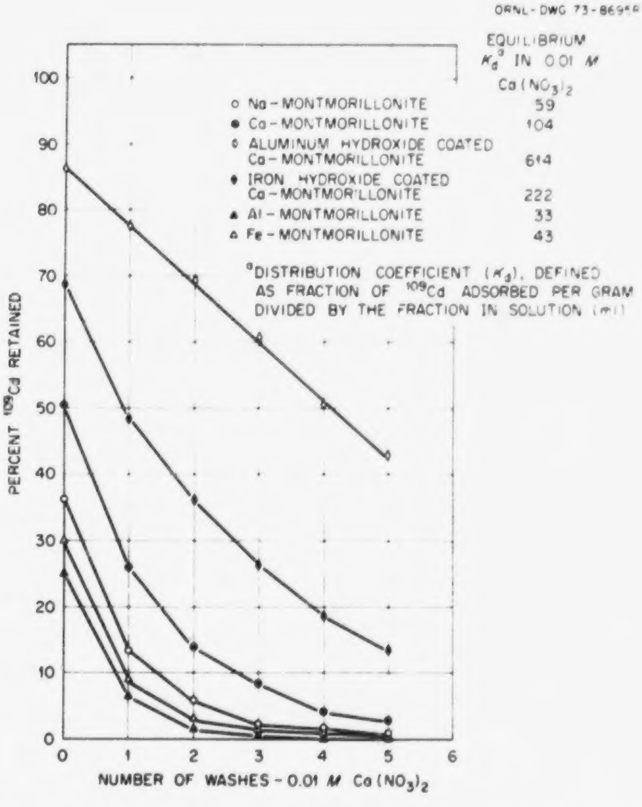


Figure 2.9. Percent ^{109}Cd retained by montmorillonites in 0.01 *M* $Ca(NO_3)_2$. Source: Brantley et al., 1974, Figure 5.2.26, p. 210.

2.3.3.2 Humus — Another important soil constituent involved in cadmium adsorption is humus, which binds very strongly to particles of clay (Bowen, 1966). This combined organo-clay fraction largely controls the adsorption of cadmium in soil (Brantley et al., 1974). The cation exchange capacity of humus depends markedly on pH (Bowen, 1966) due to the weakly acid properties of the adsorbing phenolic (⊃—O$^-$) and carboxyl (— COO$^-$) groups (Figure 2.10). Although only surface adsorption is shown, adsorption occurs

ORNL-DWG 77-5673

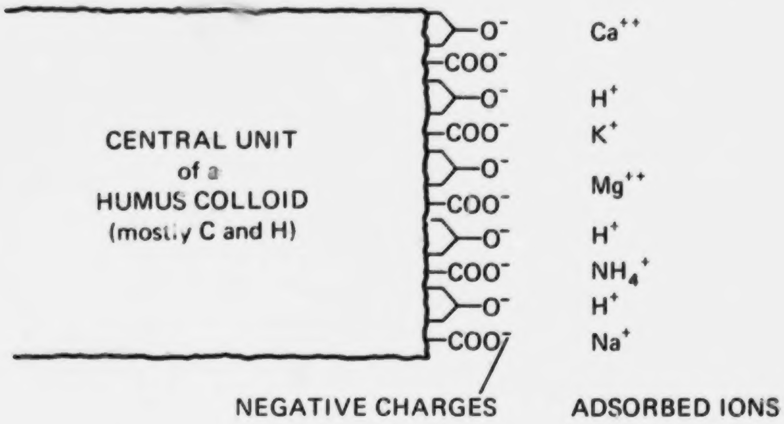


Figure 2.10. Adsorption of cations by humus colloids. Source: Brady, 1974, Figure 4.10, p. 94.

within the micelle as well (Brady, 1974). A comparison of the effect of pH on the cation exchange capacity of organic colloid humus and montmorillonite clay is shown in Figure 2.11. The exchange capacity is a measure of the negative charge, which, in turn, determines the adsorption ability of the micelle. The adsorptive capacity of humus is approximately halved as the pH is lowered from 9 to 4, whereas that of clay is almost independent of pH.

Soils become acidified by various means. For instance, release of sulfur dioxide during combustion of sulfur-containing fuel is held responsible for the high acidity of rainwater and the resultant acidification of Swedish soils (Marx, 1975). Interestingly, the deleterious effect of sulfur dioxide thus can be extended to a possible second-order effect — the release by acidification of heavy metals from soils, making them available for plant uptake (Section 4.2.1).

Three categories of humus exist: fulvic acid, humic acid, and humin. These differ in chemical and physical makeup but are similar with regard to cation adsorption and release (Brady, 1974). Consequently, laboratory adsorption studies may be facilitated by utilizing only one fraction, usually the humic or fulvic acid fraction (Gamble and Schnitzer, 1974). Bondietti et al. (1974) concluded that humic acids provide at least two types of adsorption sites, each with a different adsorption energy and coverage capacity, as shown in Figure 2.12. The total cadmium binding capacity in their test solution (i.e., the maximum concentration of complexed ion) equalled 3.58 x 10^{-4}

ORNL-DWG 77-5668

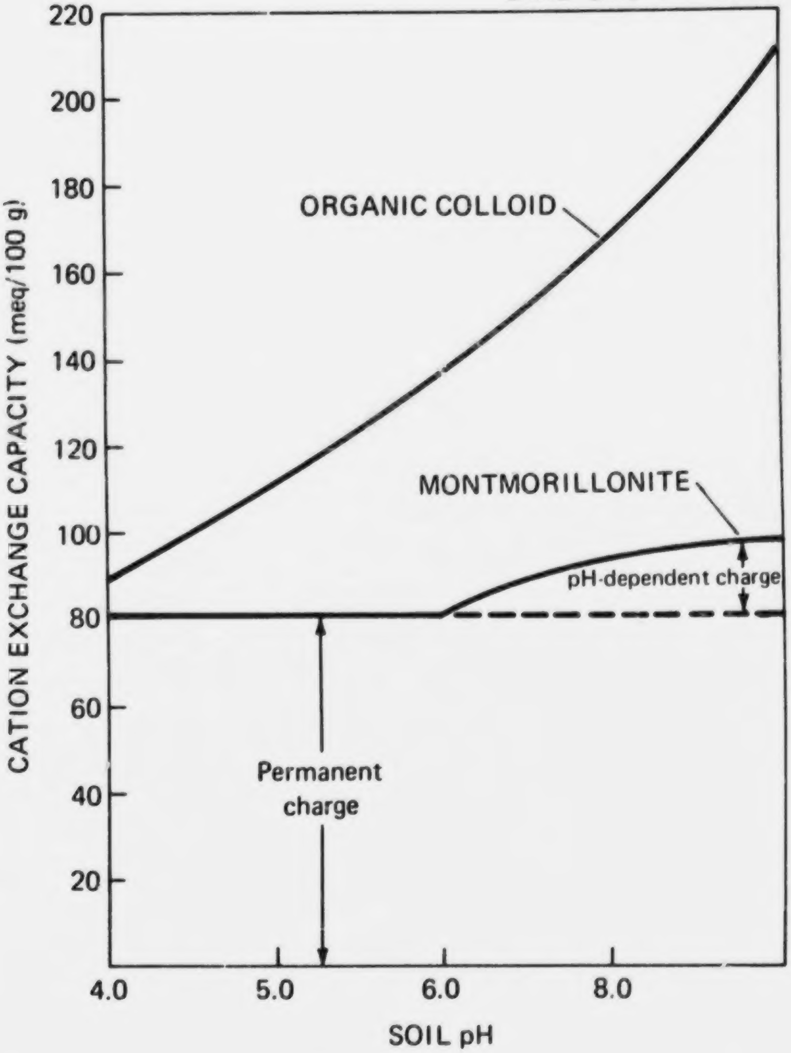


Figure 2.11. Influence of pH on the cation exchange capacity of montmorillonite and humus. Source: Brady, 1974, Figure 4.11, p. 100.

mole/liter attained at free Cd^{2+} concentrations above 10^{-3} *M*. At lower Cd^{2+} concentrations, the adsorption is very strong, although only a small portion of the available adsorption sites are occupied. As stated above, this is interpreted in terms of different adsorption sites with different affinities.

In conclusion, adsorption phenomera are involved in the behavior of cadmium in both soils (or sediments) and aqueous media. Cadmium is held most strongly by the organic-clay portion of soil, although clays coated with hydrated oxides may possess an equally high affinity for the metal. Adsorption power of humic matter is strongly pH dependent, whereas that of clay is

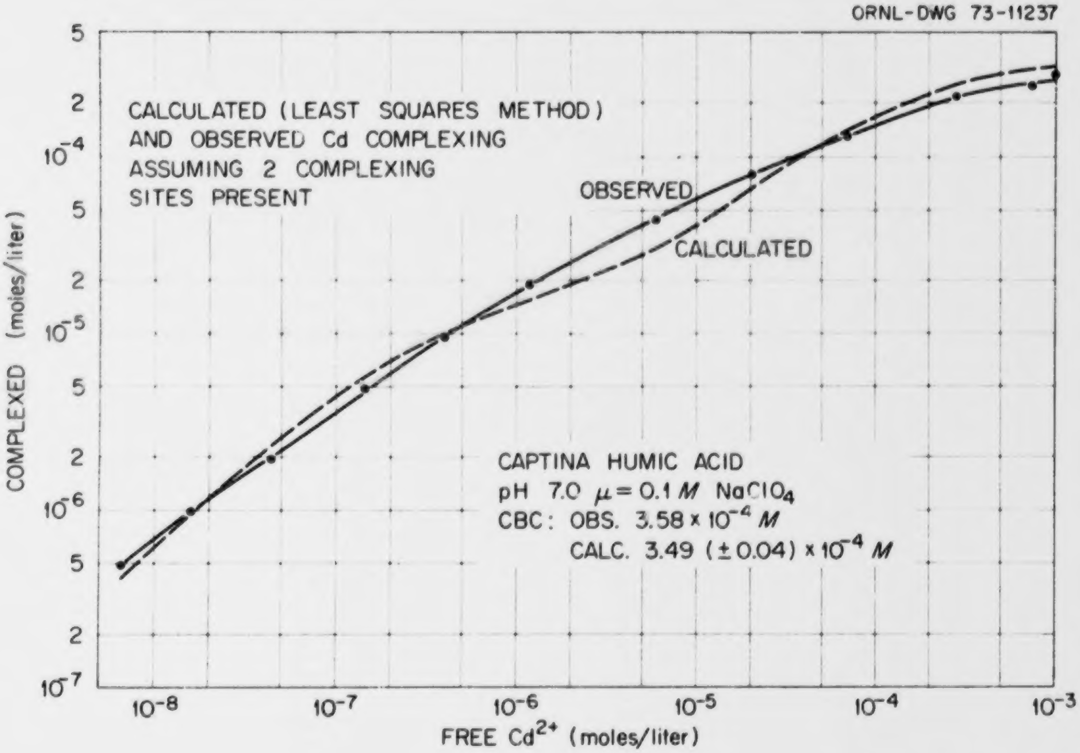


Figure 2.12. Fitting a two-site mechanism to observed complexing of Cd^{2+} by captina humic acid. Source: Bondietti et al., 1974, Figure 5.2.21, p. 190.

virtually pH independent. The role of carbonate and hydroxide (lime) in immobilizing cadmium as insoluble compounds in soil may be as important as in aqueous media.

2.3.4 Cadmium in Biological Systems

2.3.4.1 Organocadmium Compounds — Organic compounds of cadmium can be prepared in the laboratory but have not been observed in the natural environment; alkyl cadmium compounds are unstable in air and in water. Dimethylcadmium rapidly absorbs oxygen from air and is converted to a glassy mass that explodes when touched (Sheverdina and Kocheshkov, 1967). In water, dialkylcadmium sinks to the bottom of the container in the form of large drops, which are gradually converted to the hydrocarbon and cadmium hydroxide. The reaction is accompanied by a characteristic crackling noise (Sheverdina and Kocheshkov, 1967). Wood (1974), on the basis of the same approach as that used with methylmercury, concluded that cadmium is not methylated in soils and sediments; microbial methylation of cadmium has so far not been observed. Nevertheless, it is worth noting that the nephrotoxicity of methylcadmium chloride after intraperitoneal injection into cats may be greater than that of inorganic cadmium (Chang and Sprecher, 1976).

2.3.4.2 Biological Cadmium — Research into the action of cadmium in cells and tissues indicates that Cd^{2+} is the active form (Berry, Osgood, and St. John, 1974). Once within the system, cadmium may be bound to any one of a large number of soluble or structural proteins as well as to compounds of lower molecular weight. The low-molecular-weight, metal-binding proteins (metallothioneins) deserve special mention.

Methallothioneins are characterized by a high content of the sulfhydryl amino acid cysteine and of strongly bound metal ions such as cadmium and zinc (Friberg et al., 1974; Kägi and Vallee, 1960; Nordberg et al., 1972). Cadmium has a high affinity for sulfhydryl groups (Torchinskii, 1974), and as many as seven ions may be bound to each molecule of thionein. To further confirm that cadmium in metallothionein is bound via the cysteine residues, Kägi and Vallee (1960) reported a strong correlation between the light absorption by metallothionein at 250 nm and the presence of sulfhydryl groups and bound cadmium (Figure 2.13).

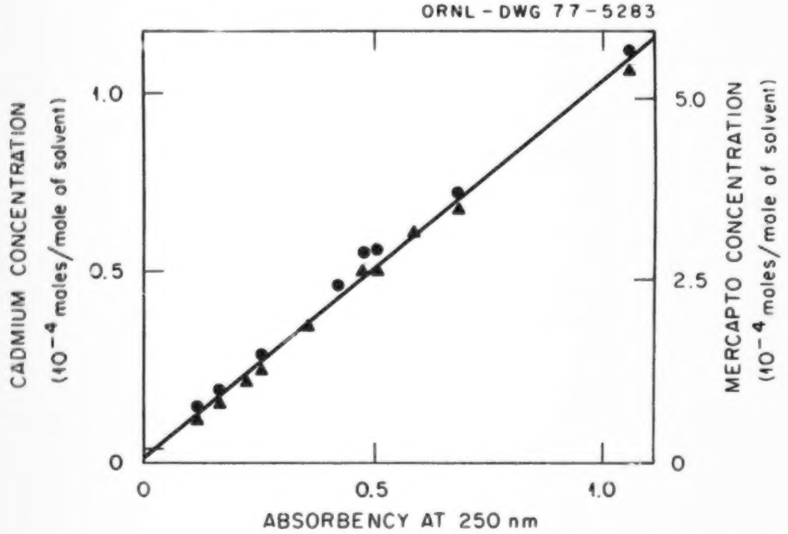


Figure 2.13. Correlation of absorbency with concentration of cadmium and of silver-titratable mercapto groups. Source: Adapted from Kägi and Vallee, 1961, Figure 2, p. 2436. Reprinted by permission of the publisher.

Metallothionein is produced especially in liver and kidney in response to exposure to several metals. The major metal ions involved are cadmium and zinc, but the function of the metalloprotein in cadmium or zinc metabolism remains in doubt. This will be discussed further in Section 6.3.1.1.

2.4 ANALYSIS FOR CADMIUM

2.4.1 Considerations in Analysis

A variety of effective methods are available for the determination of cadmium at trace-metal concentrations (Fleischer et al., 1974; Friberg, Piscator, and Nordberg, 1971; Friberg et al., 1974, 1975; Fulkerson and Goeller, 1973); progress and problems of cadmium analysis were reviewed especially by Friberg and his collaborators. No one method is best for cadmium analysis in every application; sample load, equipment availability, and cost are key considerations in method selection. Detection limits, sample matrix, specificity, analysis time, and accuracy must also be considered. These and other factors pertinent to the selection of an analytical method and the evaluation of reported analytical data are summarized here.

Sample handling and sampling techniques assume even greater importance in analytical schemes for trace metals than they do in other analytical determinations. Correctly prepared standards may be invalidated by adsorption of the metal on container walls or by leaching of contaminants from the container material. This problem is obviously aggravated by prolonged storage of such solutions before use. The composition of samples may be altered drastically by contaminated reagents used in various pretreatment procedures such as dissolution, digestion, or extraction. Care should be taken to employ only reagents of the highest purity. Even then, the quantity added should be the minimum required to accomplish the purpose so that unnecessary buildup of contaminants is avoided. A process control to monitor additions from reagents is required. Thermal processing of samples containing cadmium should be carefully controlled. Although cadmium is not as volatile as mercury or arsenic, it is, nevertheless, appreciably volatile, particularly as the chloride (Table 2.5), and caution must be exercised to avoid losses during preliminary operations with the sample.

TABLE 2.5. PERCENT RECOVERY OF METAL SALTS FROM PLATINUM CRUCIBLES AFTER HEATING AT VARIOUS TEMPERATURES FOR ONE HOUR

Metal	Sample weight (μg)	Recovery (%)								
		Chloride			Nitrate			Sulfate		
		400°C	500°C	600°C	400°C	500°C	600°C	400°C	500°C	600°C
Cadmium	10	77	56	17	102	100	83	100	100	96
Copper	20	96	96	71	104	98	77	102	100	56
Zinc	40	87	87	62	99	97	88	96	97	97
Lead	50	78	54	21	100	102	96	102	102	96

Source: Adapted from Kometani et al., 1972, Table I, p. 618. Reprinted by permission of the publisher.

2.4.2 Analytical Procedures

2.4.2.1 Preparation and Storage of Standard Solutions — Among plastic materials tested, teflon, polyethylene, and polypropylene adsorb the smallest amount of cadmium from aqueous solution, but polyethylene and polypropylene are also known to supply traces of contaminants to solutions (Shults, Lyon, and Carter, 1973, p. 428). Definitive data are needed to establish optimum sample handling and storage procedures. A safe procedure is to prepare a stock standard solution of 10^{-5} *M* or greater, using quartz or borosilicate glassware with nitric acid as a solvent. To prevent precipitation or adsorption of cadmium, the nitric acid should provide a final pH below 1 when dilutions are made of the stock solution (McFarren and Lishka, 1968). Acid-washed (10% nitric acid) and previously equilibrated glassware should be used for dilutions. Dilution flasks should always be used with solutions that have similar concentration levels. Alternatively, a stock solution of 1000 ppm cadmium in 1 *N* nitric acid may be prepared in borosilicate glassware (Sandell, 1959), and small volumes of this solution may be micropipetted directly. Stock solutions containing 1000 ppm cadmium are commercially available.

2.4.2.2 Preparation and Storage of Samples — Cadmium metal and salts are readily soluble in acid media; nitric acid is superior to hydrochloric and sulfuric acids. In contrast to zinc, alkaline media are generally not satisfactory. For organic and biological materials, a nitric acid digestion under reflux is usually adequate. This process may be followed by evaporation with perchloric or sulfuric acid, which in essence completes the oxidation of organic material, removes nitric acid, and provides a cadmium solution amenable to analysis by most methods.

Evaporation to dryness, particularly in the presence of $HClO_4$, should be avoided; explosive mixtures may be formed. Dissolution under reflux, low-temperature ashing, and freeze drying are recommended techniques for controlling solution volumes. Comparison of results using wet digestion and dry ashing techniques are shown in Table 2.6. To avoid losses by volatilization, samples should be dissolved or digested at low temperatures. It is important to collect and handle aqueous samples as carefully as dilute stock or standard solutions.

Airborne cadmium normally occurs as suspended aerosol particles — probably oxides, chlorides, and sulfates (Hise and Fulkerson, 1973, p. 206). Usually, sampling is done by collecting the particulates on a micropore filter through which a measured volume of air is passed. Collected particulates are then dissolved in acid media and subsequently handled as an aqueous solution. Alternatively, collected particulates may be analyzed directly on the filter by x-ray fluorescence (Bennett, Wagman, and Knapp, 1975).

2.4.2.3 Concentration and Separation — Analysis of samples containing cadmium in the low parts-per-billion range generally requires a procedure to concentrate the cadmium or to separate it from potential interferences, regardless of the analytical techniques subsequently employed. Simple separation procedures such as evaporation or precipitation often suffice, but more specific techniques are usually required to eliminate interfering constituents. The ideal procedure should accomplish both objectives.

2.4.2.3.1 Precipitation — Thioacetamide quantitatively precipitates the sulfides of a number of heavy metals from acid solutions. This technique is effective for solutions containing cadmium in concentrations of 5 μg/liter or more (Burrell, 1974, p. 127). Although thioacetamide precipitation

TABLE 2.6. COMPARISON OF ATOMIC ABSORPTION SPECTROSCOPY RESULTS ON NEW YORK CITY PARTICULATE SAMPLE ON FILTER PAPER PREPARED BY WET DIGESTION AND DRY ASHING

Metal	Metal content (ng/cm^2)	
	Wet digestion $HClO_4$-HNO_3	Dry ashing, 500°C
Cadmium	33, 39	22, 33
Chromium	25	25
Cobalt	N.D.,[a] <15	N.D.,[a] <15
Copper	7.6×10^2, 7.4×10^2	7.9×10^2, 7.6×10^2
Iron	46.5×10^2	49.6×10^2, 46.5×10^2
Lead	62.0×10^2, 63.6×10^2	66.7×10^2, 63.6×10^2
Manganese	42, 42	53, 64
Nickel	1.2×10^2, 1.4×10^2	1.2×10^2, 1.2×10^2
Vandium	3.7×10^2	4.3×10^2
Zinc	2.17×10^2, 2.17×10^2	21.7×10^2, 21.7×10^2

[a] N.D. — not detected.

Source: Adapted from Kometani et al., 1972, Table IV, p. 618. Reprinted by permission of the publisher.

increases sensitivity and eliminates matrix effects, the technique is not widely used because of the excessive time and attention required. (Note: Thioacetamide is listed by the National Institute for Occupational Safety and Health as a possible carcinogen.)

2.4.2.3.2 Solvent extraction — Liquid-liquid solvent extraction is probably the single most useful separation method for all chemical analyses (Andelman, 1971, p. 39). Its versatility is exemplified by the variety of its applications with cadmium. Cadmium can be extracted from aqueous into organic media either as an ion-association or a chelate complex. In general, ion-association complexes are used for milligram and larger quantities of cadmium, while chelate complexes are used for trace quantities.

Quaternary ammonium salts of long chain amines, when dissolved in an organic solvent, can extract a variety of metals as anionic-association complexes. Cadmium can be extracted quantitatively by tetrahexylammonium iodide into methyl isobutyl ketone from acidic solutions. Cadmium is readily stripped from the organic phase with the appropriate aqueous solution, thus providing a convenient technique for concentration and separation of the metal from a variety of environmental media (Maeck et al., 1961).

Extraction with dithizone (diphenylthiocarbazone) is probably the most widely used separation technique for cadmium. Typically, the cadmium dithizonate is extracted into carbon tetrachloride or chloroform from an alkaline aqueous medium containing suitable metal buffers and masking agents. Cadmium is them recovered for analysis from the organic phase with strong acid (Sandell, 1959, p. 355).

Under conditions similar to those cited above, cadmium can be extracted into carbon tetrachloride as the diethyldithiocarbamate (Sandell, 1959, p. 352). In alkaline solution, metals which form sufficiently strong complexes with cyanide ions remain in the aqueous phase. Similarly, cadmium can be extracted into methyl isobutyl ketone as the pyrrolidine dithiocarbamate (Yeager, Cholak, and Meiners, 1973). The latter solvent is compatible with flame atomic absorption spectrographic techniques.

2.4.2.3.3 Chromatographic methods — Numerous chromatographic separation methods have been applied to cadmium in recent years. Alumina, paper, cation and anion exchange resins, and chelating agents, as well as zinc sulfide, have been used. The ion exchange resin technique is particularly convenient. For example, Dowex-1 (an anion exchange resin) sorbs cadmium, zinc, and bismuth from a solution of 0.1 *M* HCl and 10% NaCl, but rejects iron(III), copper, manganese, aluminum, nickel, cobalt, chromium(III), rare earths, titanium, beryllium, and the alkaline earth ions. Washing the resin with a solution of 2 *M* NaOH and 2% NaCl solution removes zinc, lead, and some bismuth. The cadmium and remaining bismuth can be removed with 1 *M* HNO_3 (Kolthoff and Elving, 1961, p. 192). Collection and concentration of trace elements on ion exchange resins in the field and subsequent elution and analysis in the laboratory preserve the sample while avoiding the adsorptive losses or contamination that can occur during storage. This technique deserves greater consideration than it is presently given (Environmental Instrumentation Group, 1973*a*).

2.4.2.3.4 Electrodeposition — One of the most convenient methods for effecting the separation of cadmium from other metals is electrolytic deposition on either a platinum or mercury electrode. The electrolysis can be performed in neutral, weakly acid, or alkaline media. The completeness of the separation depends on the difference between the reduction potentials of the metals to be separated. To assure the quantitative separation of cadmium from such metals as zinc, copper, and lead, it is advisable to perform the electrolysis at an electrode whose potential is controlled at an optimum value. For example, cadmium can be quantitatively separated from zinc by an electrodeposition from sulfuric or hydrochloric acid at a mercury electrode that is controlled at -0.85 V versus a saturated calomel reference electrode (Kolthoff and Elving, 1961, p. 184). Cadmium is collected as an amalgam which can be back-electrolyzed at a more positive potential to achieve greater purity, if desired (Section 2.4.2.4.3). Electrodeposition can be used with pyrolytic graphite as the working electrode, followed by neutron activation analysis, or with platinum, gold, or copper electrodes for spark-source spectrometric analysis.

2.4.2.4 Methods of Analysis — Cadmium can be determined by a variety of analytical procedures. Those which are currently important or show future promise are described in this section. Emphasis is placed on performance and limitations of each procedure rather than on minute details. Table 2.7 lists common instrumental methods for the analysis of cadmium. Sensitivity, precision, accuracy, and optimum concentration ranges of samples vary not only among different methods, but also among various models of particular instruments. The tabulated data must therefore be considered representative rather than definitive.

Several of the terms used in Table 2.7 are defined variously in the literature. Sensitivity (Column 4) denotes the smallest quantity that can be determined reliably by the designated technique or instrumentation. In analytical methods utilizing continuous sampling, such as atomic absorption spectrometry, sensitivity usually means the concentration of analyte that yields an absorption of 1% or an absorbance of 0.0044. These data cannot be compared with sensitivities given for methods using noncontinuous sampling techniques, such as flameless atomic absorption spectrometry (Burrell, 1974, p. 76). Column 6 gives the precision of each method as relative standard deviation (i.e., the standard deviation of a set of samples expressed as a percentage of the mean). It also indicates sample concentration because the reported precisions of trace-level analyses strongly depend on sample size. Column 7 records accuracies of various methods in terms of percentage recovery from spiked samples. Literature values for the accuracy of a given method vary widely, and only a few interlaboratory or interprocedural comparisons are available. In general, however, only conservative literature reports are cited; under ideal conditions, even more favorable results may be expected.

2.4.2.4.1 Spectrophotometric analysis — Spectrophotometric procedures are based on measuring light absorbed by a sample at a particular wavelength. The most popular method for determining trace levels of cadmium has been extraction with dithizone followed by a spectrophotometric reading (American Public Health Association, American Water Works Association, and Water Pollution Control Federation, 1971, p. 77). Although tedious and time-consuming, this technique can be both sensitive and specific. The extraction procedure depends on the type of sample being processed as well as the type of matrix and interferences that might be present. In general, extraction is performed at pH greater than 12 with cyanide and/or tartrate present to prevent hydrolysis of the cadmium and to control precipitation and extraction of other metals. The dithizone-chloroform extract is washed first with dilute sodium hydroxide, then water, and the optical absorbance of the cadmium dithizonate is measured at 520 nm. Lead, zinc, and bismuth are not extracted and do not interfere. Accurate results depend on the use of high-purity dithizone and chloroform. As an alternative procedure, the cadmium dithizonate is acidified with 0.5 *M* HCl, and the color intensity of liberated dithizone is measured at 620 nm.

The dithizone method is useful for processing a variety of liquid and solid samples, especially when preconcentration is needed or when high levels of other cations may be present. It is now being superseded, however, by the more sensitive and convenient technique of atomic absorption spectrometry.

TABLE 2.7. COMMONLY USED INSTRUMENTAL METHODS FOR THE ANALYSIS OF CADMIUM

Analytical method	Important application	State of measured cadmium	Sensitivity	Interfering substances	Precision		Accuracy	
					Relative standard deviation (%)	Sample concentration	Recovery from spiked sample (%)	Sample concentration
Spectrophotometry	Aqueous samples, biological materials	Cadmium dithizone complex	50 µg/liter	Concentrations of metal ions normally present in natural waters do not interfere.[b]	±25	50 µg/liter[b]	94	50 µg/liter[b]
Polarography	Metal-bearing surface waters	Solvated ion	1000 µg/liter (DC)	Dependent on metal ion activity	±3	1 µg/g (DC)	97	0.12 µg/g (with tenfold preconcentration)
			4 µg/liter (pulse)		±2 (pulse)	100 µg/liter	58	0.12 µg/g (no preconcentration)
Emission spectroscopy	Ores, alloys, solids	Vibrationally excited atoms	20 µg/g	Chemical matrix alters the line intensity and background.	19	220 µg/g	90	1000 ppm
Atomic absorption spectrometry (flame)	Biological material, effluents, saline and fresh waters	Free unexcited atoms	10 µg/liter (direct) 1 µg/liter (extracted)	Silicon and sodium chloride interfere under some conditions.	±30	71 µg/liter	98.6	71 µg/liter[b]
Atomic absorption spectrometry (flameless)	Biological materials, effluents, saline and fresh water	Free unexcited atoms	0.25 µg/liter	Charring of organic material and inorganic salts such as NaCl in the matrix leads to high cadmium readings. Excess HCl may result in loss of volatile $CdCl_2$	±47 (urine) ±8	1.5 µg/liter, 8.8 µg/liter	95	10-60 ng/g
Neutron activation analysis	All media and materials, biologic and inorganic	As is	0.1 µg/g (pure water) 50 µg/liter (river water)	X rays from many elements may overlap energy peaks from cadmium (e.g., ^{24}Na and ^{38}Cl in seawater samples). Compton background may limit sensitivity.	±10	0.5 µg/liter	20	50

(continued)

TABLE 2.7 (continued)

Analytical method	Important application	State of measured cadmium	Sensitivity	Interfering substances	Precision		Accuracy	
					Relative standard deviation (%)	Sample concentration	Recovery from spiked sample (%)	Sample concentration
Anodic stripping voltammetry	Ores, alloys, dusts, industrial wastes, saline and surface waters, biological materials	Amalgam	0.1 μg/liter[l]	Substances normally present in naturral waters do not interfere.	±4.5	0.1 μg/g[m]	90	1 μg/liter[l]
X-ray fluorescence	Air particulate samples	As is	0.006 μg/cm²[n]	Line interferences not significant with high-resolution wavelength-dispersive spectrometer.	±12	40 μg/cm²[o]	95	40 μg/cm²[o]

Sources:

[a] Environmental Instrumentation Group, 1973*a*.

[b] American Public Health Association, American Water Works Association, and Water Pollution Control Federation, 1971, pp. 77, 80.

[c] Environmental Instrumentation Group, 1973*b*.

[d] Shults, Lyon, and Carter, 1973, pp. 433, 435.

[e] Osteryoung and Osteryoung, 1972.

[f] Coleman, Van Atta, and Klatt, 1972.

[g] Friberg et al., 1974.

[h] U.S. Environmental Protection Agency, 1974, p. 102.

[i] Ross and Gonzalez, 1974, p. 446.

[j] Kjellström et al., 1974, p. 98.

[k] Weiss et al., 1974, p. 176.

[l] Zirino and Healey, 1972, p. 243.

[m] MacLeod and Lee, 1973.

[n] Bennett, Wagman, and Knapp, 1975.

[o] Camp et al., 1975.

2.4.2.4.2 Spectroscopic methods — These useful methods for determining cadmium include emission spectroscopy, flame and flameless spectrometry, and spark-source mass spectrometry.

Emission spectroscopy has been used a great deal for rapid qualitative or semiquantitative analysis of cadmium in a variety of liquid and solid sample forms, especially metals, alloys, ores, and homogeneous solids which require multielement determinations (Environmental Instrumentation Group, 1973*a*, p. 14). Liquid samples are usually deposited on metal or graphite electrodes and dried before analysis. Atoms or atomic ions form by excitation of the sample in an electrical arc, spark, or plasma. The energy of the excited atoms or ions is emitted as discrete radiation or as bands of radiation for molecular forms. Spectra consist of sharp lines for which the wavelength and intensity are characteristic of the element. Comparison of spectra of known and unknown materials provides qualitative identification of elements in the sample. For quantitative analysis, the usual practice is to measure the ratios of line intensities of the element under study and compare them to those of a known element. The known element may be part of the sample or may be added to the sample and thus be subjected to the same conditions as the element of interest. This minimizes the influence of procedural variables. For accurate work the ratios are compared with standards prepared by the same techniques. Accuracy is about 10% if the standard addition technique is used and the concentration is approximately 0.1% (Environmental Instrumentation Group, 1973*a*, p. 14). Emission spectroscopy using plasma excitation of the sample is more sensitive than conventional direct or alternating current arc or spark sources (Environmental Instrumentation Group, 1973*b*, p. 6).

Flames can be used to volatilize, atomize, or excite many elements, which can then be determined by the amount of light emitted from or absorbed by these elements in selected zones of the flame (flame spectrometry). Cadmium can be determined by atomic emission, atomic absorption, and atomic fluorescence modes of operation. Usual detection limits reported for these techniques are 50, 10, and 0.2 μg/liter respectively (Shults, Lyon, and Carter, 1973, p. 435). Atomic absorption is most often used and is rapidly becoming the accepted method for determining cadmium at the trace level. Though not as sensitive as anodic stripping voltammetry, this method has the definite advantage of being faster and less tedious. The cadmium lines at 228.8 nm and 326.1 nm are usable; the 228.8-nm line is more intense and preferred for analysis. Detection limits can be improved either by using a long optical (flame) path length or by preconcentration. The former approach has been reported to provide detection limits under 1 μg/liter (Environmental Instrumentation Group, 1973*a*, p. 14). The latter approach — extraction plus direct aspiration of the extract into the flame — provides detection limits comparable to those of anodic stripping techniques. The atomic absorption technique is useful for all types of samples, particularly trace-level determinations of cadmium in fresh and marine waters, industrial effluents, and biological materials. The atomic fluorescence technique needs further development to gain wide acceptance in the determination of trace levels of cadmium in environmental samples.

Conventional flame spectrometry techniques normally convert 2% to 8% of the sample to the atomic state (Environmental Instrumentation Group, 1973*a*,

p. 5). In some instances, efficiency and sensitivity can be improved by replacing the flame with a carbon, graphite, or tantalum-ribbon furnace in which the sample is sequentially dried, ashed (if necessary), and heated to incandescence (flameless spectrometry). Smoke or salt particles produced during the heating steps can cause scattering and attenuation of light intensities. In addition, flameless techniques generally produce an absorbance peak of relatively short duration compared to the "steady" signal obtained from a flame. Nevertheless, direct analysis of cadmium at the level of 1 ng/liter is now feasible for some samples (Environmental Instrumentation Group, 1973*a*, p. 14), and rapid extension of the technique to new types of samples continues. The flameless atomic absorption technique may be used to analyze all types of samples; however, it is probably most effective for the direct analysis of solid biological and environmental samples without prior processing.

Cadmium may be determined by exciting the sample with a radio-frequency spark, followed by separation of the resulting cadmium ions according to their mass (spark-source mass spectrometry). This technique is applicable to virtually any matrix, but results are only semiquantitative. The method can be accurate within ±5%, however, when used with an isotope dilution technique (Shults, Lyon, and Carter, 1973, p. 435). In this procedure, ^{106}Cd is added to the sample, and isotopic equilibrium is established between the added ^{106}Cd and the "normal" cadmium in the sample. In most cases, isotopic equilibrium is easily attained by refluxing the sample with a perchloric acid–nitric acid mixture. This treatment also serves to oxidize any organic matter present in the sample. Once isotopic equilibrium is reached, any technique that permits the transfer of 1 to 10 ng of cadmium from the solution to the surface of a suitable substrate (electrode) may be used. Solvent extraction may be employed, if necessary, to concentrate the cadmium before transfer to the substrate. Extraction with dithizone is suitable. High extraction efficiencies are not required since the analysis at this point depends only on establishing a ratio between two isotopes — for example, ^{114}Cd and ^{106}Cd. This procedure is more time-consuming and expensive than some of the methods discussed previously; however, it is virtually free from matrix effects and has the advantage of allowing analysis for many elements simultaneously. When the isotope dilution technique is used, the method has a detection limit of about 1 μg cadmium per liter. It is attractive for samples requiring multielement determinations at low trace-element concentrations.

2.4.2.4.3 Electrochemical techniques — Cadmium can be determined by a variety of electrochemical techniques. The most popular methods are polarography and anodic stripping voltammetry. In polarography, the current-voltage curve for electrolysis at a small mercury drop is obtained. The curve is sigmoidal: height reflects the concentration of electroactive material present and location (potential) reflects the identity of the material. Conventional polarography can easily be used to determine cadmium at the 1 μg/g level, with precision and accuracy of approximately ±3%. With sophisticated instrumentation, lower concentrations can be analyzed (Shults, Lyon, and Carter, 1973, p. 433). For example, 0.1 μg/g solutions can be analyzed by "derivative" polarography, and 0.01 μg/g solutions can be analyzed by "pulse" polarography. Conventional polarography techniques are useful in analyzing metal-bearing liquid wastes and effluents and surface water samples containing relatively high concentrations of cadmium. Natural water samples and other environmental liquids containing cadmium in the parts-per-billion range cannot be analyzed without preconcentration. The latter samples may, however, be satisfactorily processed by "derivative" and "pulsed" polarographic techniques. This technique differs from ordinary polarography because the current is measured twice during each drop, and the difference is recorded. Well-defined signals are obtained from concentrations of cadmium as small as 4 ppb (Osteryoung and Osteryoung, 1972).

Anodic stripping voltammetry is a sensitive electrochemical technique for determining cadmium. Cadmium is electrodeposited under strictly controlled experimental conditions (e.g., stirring rate and electrode potential), and the resulting cadmium amalgam is electrolytically dissolved to determine the amount of cadmium electrodeposited. The dissolution or "stripping" step can be accomplished in several ways. Voltammetric stripping involves the measurement of stripping current as a function of electrode potential, whereas coulometric stripping measures the amount of charge required to dissolve the amalgam. In either mode of operation, solutions containing 1 μg cadmium per liter can be analyzed with an accuracy of about ±10% (Shults, Lyon, and Carter, 1973, p. 433). With care, this technique can be used routinely. Zinc and cadmium-to-zinc ratios can also be determined. Because of great sensitivity, the anodic stripping voltammetry technique can be used to analyze most freshwater samples without preconcentration. With suitable sample preparation, this technique is also useful for industrial wastes, biological materials, dusts, alloys, and ores.

In recent years, ion-selective electrodes have been developed which respond to various cations and anions. Such an electrode is available for measuring cadmium concentration. Although sensitivity is not sufficient for monitoring cadmium in natural waters, it may be suitable for monitoring cadmium in waste materials and other special situations. Semilogarithmic response extends down to about 1 μg/g cadmium (Shults, Lyon, and Carter, 1973, p. 434). Interference by copper, lead, silver, and mercury may be severe, and since only the activity of the free metal ion is measured, any complexed metal will go undetected.

2.4.2.4.4 Neutron activation analysis — Neutron activation analysis is one of the most sensitive modern analytical techniques for the determination of trace elements. Samples and known standards are irradiated in a nuclear reactor during which time neutrons are captured by various nuclides in the sample. Radioactive isotopes are usually produced, making it possible, by appropriate measurement techniques, to identify the daughter activities and relate them to the parent isotope. By comparison with the activity induced in the standards, the amount of sought isotope can be calculated. The induced activity, and hence the sensitivity for determining the parent nuclide, is proportional to the neutron flux, to the nuclear cross section of the atoms, and to the amount of the parent isotope present. Neutron fluxes of 10^{12} to 10^{14} per square centimeter per second are easily available in modern reactors; therefore, for reasonable length irradiations (a few seconds to a few days) most elements can be determined at the level of 10^{-8} to 10^{-10} g (Shults, Lyon, and Carter, 1973, p. 437). The commonly used reaction for activation analysis is $^{114}Cd(n, \gamma)^{115}Cd$. Since ^{114}Cd has a cross section of 1.1×10^{-24} cm^2 and produces a 53.5-hr half-life decay for ^{115}Cd, which has only 25% branching through a measurable gamma ray, sensitivity for nondestructive neutron activation analysis of cadmium is not as great as for some other elements. Cadmium can be determined nondestructively at the level of 10^{-7} to 10^{-9} g in matrices that do not activate significantly. Chemical separation of cadmium allows sensitivities several orders of magnitude greater than this. The technique is applicable to all types of samples, usually in their existing state. Aqueous samples may require concentration by evaporation, precipitation, solvent extraction, electrodeposition, or ion exchange. Minimum detectable concentrations of cadmium are relatively high for river water (0.05 ppm) and seawater (16 ppm) because of interference from major constituents, such as sodium (Environmental Instrumentation Group, 1973*a*, p. 15).

The determination of cadmium by neutron activation analysis is relatively expensive; it is economically competitive with other techniques only when nondestructive methods must be applied or when several elements are determined simultaneously, thus reducing the analytical cost per element.

2.4.2.4.5 X-ray fluorescence — Recent developments in x-ray sources and instrumentation have resulted in increased acceptance of x-ray fluorescence as an analytical method for the simultaneous detection of several elements in a wide range of samples. X-ray fluorescence may be induced by excitation sources such as electron beams, high-energy x rays, or alpha, beta, and gamma radiation from radioisotope sources. When the sample is irradiated, a series of characteristic x-ray lines are then emitted as the ejected electron is replaced by an electron from an outer orbital of the excited atom. Two techniques are used to resolve the spectra. Wavelength-dispersive spectrometers employ analyzing crystals to resolve the fluorescence spectra. Alternatively, energy-dispersive spectrometers utilize solid-state detectors in conjunction with multichannel analyzers.

X-ray fluorescence has not been the best method for determining cadmium in trace-level samples. Cadmium is only poorly excited by conventional x-ray sources; thus, a sensitivity of a few parts per million is the best that can be expected. Watanabe, Berman, and Russell (1972) stated that cadmium cannot be determined simultaneously with other elements because special targets and analyzing crystals are required. However, Bennett, Wagman, and Knapp (1975) analyzed air pollution particulate samples collected on membrane filters for cadmium. A spectrometer system with a 16-crystal monochromator was capable of determining up to 30 elements simultaneously. The detection limit for cadmium was 6 μg/cm^2 and was attributed to a combination of low background and efficient excitation of the cadmium K_α line by a chromium target x-ray tube. The method is nondestructive, requires no pretreatment, and may be completed with nominal counting times of 100 sec.

2.4.3 Comparison of Analytical Methods

Until the last decade, the spectrophotometric method utilizing dithizone was the most widely used technique for determining cadmium. As discussed above, the method must be combined with extraction procedures to achieve adequate sensitivity and specificity. During the last few years, however, this method has been largely replaced by atomic absorption spectrometry. The latter method usually requires extraction of cadmium as the ammonium pyrrolidine dithiocarbamate complex into methyl isobutyl ketone, followed by aspiration into an air-acetylene flame. However, many aqueous samples containing 25 to 250 μg cadmium per liter can be analyzed satisfactorily without pretreatment. The spectrophotometric method using dithizone is neither sensitive nor specific for measuring cadmium in natural water samples, although it can be made so by extraction methods.

The chief need in analysis of environmental samples of cadmium is greater sensitivity so that sample pretreatment can be minimized or eliminated. Fortunately, several such techniques are emerging from developmental laboratories and are becoming generally available. Flameless atomic absorption spectrometers equipped with a graphite furnace, a carbon rod, or a tantalum ribbon rather than a flame may extend the detection limit of cadmium by a factor of 10 to 1000 (Environmental Instrumentation Group, 1973*b*, p. 16). Direct analysis of water and biological samples is feasible with minimal pretreatment and greatly simplifies the procedure. The anodic stripping voltammetry technique can often determine cadmium in the low parts-per-billion range without sample pretreatment. Differential pulse polarography is 100 to 1000 times more sensitive than classical polarography, permitting analysis of cadmium at the level of 1 to 100 ppb. Preparation of biological samples for atomic absorption spectrometry was described by Gross et al. (1975) and special solutions for analyzing blood by anodic stripping voltammetry have been reported (Environmental Science Associates, Inc.).

Two somewhat older analytical techniques are available for analyses of cadmium in the parts-per-billion range: spark-source mass spectrometry with isotope dilution and neutron activation analysis. These established methods are not, in general, economically competitive with the methods described above, unless simultaneous determinations of a number of other elements are required.

The x-ray fluorescence method allows multielement analysis, requires minimal sample handling, and is essentially nondestructive. The technique is useful in many applications such as particulate samples collected from ambient air and source emission streams. However, low sensitivity and general inconvenience prevent x-ray fluorescence from being the best method for determining cadmium at trace levels (Watanabe, Berman, and Russell, 1972).

2.4.3.1 Standardization — Trace-level determinations of cadmium are required in an extensive variety of samples: biological specimens such as plants, animal tissues, bone, and blood; soils and sediments; geological specimens; industrial wastes and products, including sludges, dusts, and filter deposits; metals and alloys; paints and pigments; plastics; and various fresh and marine water supplies. Reliable analyses require (1) standard procedures for collection, preparation, and storage of these specimen types and (2) standard methods for concentrating or separating the contained cadmium for determination by one of several analytical techniques. Standardization, however, is only beginning; earliest efforts have been directed toward development of procedures for groundwater, surface water, domestic and industrial waste effluents, and treatment process samples. Thus, *Standard Methods for the Examination of Water and Wastewater*, 13th ed., 1971, published jointly by the American Public Health Association, the American Water Works Association, and the Water Pollution Control Federation, prescribes standard sample handling techniques and analytical procedures for many metals, including spectrophotometric and atomic absorption techniques for cadmium at the trace level. More recently, the U.S. Environmental Protection Agency published the *Handbook for Analytical Quality Control in Water and Wastewater Laboratories*, 1972, defining standards useful in many aspects of the work. In a companion volume, *Manual of Methods for Chemical Analysis of Water and Wastes*, 1974, the U.S. Environmental Protection Agency established standard procedures for determining many constituents of water samples, including the analysis of trace levels of cadmium by atomic absorption spectrometry. Much work remains to be done in this area. One of the most pressing needs is to make standard materials representative of environmental samples more available. Secondary standards, based on analysis rather than synthesis, should be available for comparison of procedures as well as for interlaboratory comparisons.

2.4.3.2 Interlaboratory Comparison — Relatively few interlaboratory comparisons of cadmium analyses at the trace level have been reported. In one study, conducted by the Quality Assurance Laboratory Branch of the Methods Development and Quality Assurance Research Laboratory, Cincinnati, Ohio, six synthetic concentrates containing varying levels of aluminum, cadmium, chromium, iron, manganese, lead, and zinc were added to natural water samples (U.S. Environmental Protection Agency, 1974). Samples were distributed to various laboratories for analysis by atomic absorption spectrometry. The statistical results for cadmium detection are given in Table 2.8. The precision and accuracy obtained among laboratories established the adequacy of the atomic absorption spectrometry method for cadmium down to 1 ppb for sample types described.

TABLE 2.8. INTERLABORATORY STUDY ON TRACE LEVEL ANALYSIS OF CADMIUM BY ATOMIC ABSORPTION SPECTROMETRY

Number of labs	True value (μg/liter)	Mean value (μg/liter)	Standard deviation (μg/liter)	Accuracy (% bias)
74	71	70	21	-2.2
73	78	74	18	-5.7
63	14	16.8	11.0	19.8
68	18	18.3	10.3	1.9
55	1.4	3.3	5.0	135
51	2.8	2.9	2.8	4.7

Source: U.S. Environmental Protection Agency, 1974, p. 102.

In an earlier study (McFarren and Lishka, 1968), the Analytical Reference Service, Cincinnati, Ohio, prepared water samples having the composition given in Table 2.9. The samples were distributed to 79 participating laboratories for analysis of nine designated metals by procedures specified in *Standard Methods for the Examination of Water and Wastewater*, 12th ed. For analysis of cadmium, the indicated methods were spectrophotometry, using dithizone, and polarography. The results for cadmium are listed in Table 2.10. The dithizone method, giving poor precision but excellent accuracy, is obviously superior on both counts to the polarographic technique. However, both methods suffer when compared with the more modern atomic absorption technique previously described.

TABLE 2.9. COMPOSITION OF WATER METALS SAMPLE USED IN INTERLABORATORY COMPARISON

Compound	Metal	Concentration (mg/liter)
$K_2Al_2(SO_4)_4 \cdot 24H_2O$	Al	0.50
Copper metal	Cu	0.47
$Fe(NH_4)_2(SO_4)_2 \cdot 6H_2O$	Fe	0.30
$KMnO_4$	Mn	0.12
Zinc metal	Zn	0.65
$AgNO_3$	Ag	0.15
Cadmium metal	Cd	0.05
$K_2Cr_2O_7$	Cr	0.11
$Pb(NO_3)_2$	Pb	0.07

Source: Adapted from McFarren and Lishka, 1968, Table I, p. 254. Reprinted by permission of the publisher.

In a study by the U.S. Environmental Protection Agency in which trace elements in fuels were monitored (von Lehmden, Jungers, and Lee, 1974), nine laboratories using similar analytical methods were asked to determine the concentration of 28 elements, including cadmium, in the same fuel as well as in fly-ash matrices. The analytical methods used included neutron activation analysis, atomic absorption spectrometry, spark-source mass spectrometry, optical emission spectrometry, anodic stripping voltammetry, and x-ray fluorescence. The cadmium results are listed in Table 2.11. The wide range of concentrations illustrates the difficulties in determining trace elements in different matrices and again emphasizes the need for standard reference materials certified in trace-element concentrations.

TABLE 2.10. SUMMARY OF INTERLABORATORY DATA ON MANGANESE, SILVER, AND CADMIUM ANALYSES OF WATER SAMPLES

Method of analysis	Number of analyses	Mean (mg/liter)	Standard deviation (mg/liter)	Mean error (mg/liter)	Relative standard deviation (%)	Relative error (%)
			Manganese, 0.12 mg/liter			
Persulfate[a]	33	0.118	0.031	0.00	26.3	0.0
Periodate[a]	14	0.150	0.054	0.03	36.0	25.0
Spectrograph	4	0.113	0.022	0.01	19.4	8.3
Atomic absorption	3	0.127	0.025	0.01	19.6	8.3
Formaldoxime	3	0.180	0.069	0.06	38.4	50.0
			Silver, 0.15 mg/liter			
Dithizone[a]	14	0.049	0.030	0.00	61.0	66.6
Spectrograph	3	0.090	0.066	0.06	73.5	40.0
			Cadmium, 0.05 mg/liter			
Dithizone[a]	44	0.053	0.013	0.00	24.5	0.0
Polarograph	4	0.040	0.034	0.01	68.0	20.0

[a] Standard method.

Source: Adapted from McFarren and Lishka, 1968, Table V, p. 258. Reprinted by permission of the publisher.

TABLE 2.11. COMPARISON OF CADMIUM CONCENTRATIONS IN COAL, FLY ASH, FUEL OIL, AND GASOLINE BY ANALYTICAL METHOD AND BY LABORATORY

Sample matrix	Analytical method	Laboratory code[a]	Sample code[b]	Analysis	
				(μg/g)	(μg/ml)
Coal	Spark-source mass spectrometry[c]	1		6.	
	Spark-source mass spectrometry[c]	3		<1.	
	Spark-source mass spectrometry[c]	6		0.7	
	Optical emission spectrometry[d]	1		<30	
	Optical emission spectrometry[d]	3		<10	
	Nuclear activation analysis (instrumental)	3		<3	
	Nuclear activation analysis (instrumental)	4		<40	
Fly ash	Spark-source mass spectrometry[c]	1		<3	
	Spark-source mass spectrometry[c, e]	1		<6	
	Spark-source mass spectrometry[c]	3		2	
	Spark-source mass spectrometry[c]	6		2.3	
	Optical emission spectrometry[d]	1		<50	
	Optical emission spectrometry[d, e]	1		<100	
	Neutron activation analysis (instrumental)	4		<90	
Fuel oil (No. 6)	Spark-source mass spectrometry[f]	1			0.003
	Spark-source mass spectrometry[g]	3			0.83
	Optical emission spectrometry[f]	1			0.4
	Optical emission spectrometry[g]	1			1.
	Atomic absorption spectrometry	1			<0.2
Premium gasoline	Spark-source mass spectrometry[h]	1	G-10		0.001
	Optical emission spectrometry[i]	1	G-10		<0.1
	Optical emission spectrometry[d]	1	G-10		<1.
	Neutron activation analysis (instrumental)	4	G-10		<20
	Atomic absorption spectrometry	1	G-10		<0.15
	Spark-source mass spectrometry	3	G-1		0.023
	Anodic stripping voltammetry[h]	7	G-1		0.006

[a] The nine participating laboratories were coded to maintain anonymity.

[b] Comparison includes two samples (G-1 and G-10) of the same brand name collected from the pumps of two different retail service stations.

[c] Analysis on sample direct.

[d] Direct current arc on sample direct.

[e] Duplicate sample submitted for spark-source mass spectrometry and optical emission spectrometry analysis only.

[f] 450°C ashing followed by ash analysis.

[g] H_2SO_4–HNO_3 dissolution, dried, and analyzed.

[h] HCl extraction preparation.

[i] Analysis of residue after combustion of sample.

Source: Adapted from von Lehmden, Jungers, and Lee, Tables I-IV, pp. 240-243. Reprinted by permission of the publisher.

SECTION 2

REFERENCES

1. American Public Health Association, American Water Works Association, and Water Pollution Control Federation. 1971. Standard Methods for the Examination of Water and Wastewater, 13th ed. American Public Health Association, Washington, D.C. 874 pp.

2. Andelman, J. B. 1971. Concentration and Separation Techniques. In: Instrumental Analysis for Water Pollution Control, K. H. Mancy, ed. Ann Arbor Science Publishers, Inc., Ann Arbor, Mich. pp. 33-60.

3. Baes, C. F., Jr. 1973. The Properties of Cadmium. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 29-54.

4. Bailey, S. M., G. R. Helz, and R. L. Harris. 1975. Investigation of the Transport of Metals and Orthophosphate Away from a Sewage Treatment Plant Outfall. Environ. Lett. 10(2):159-169.

5. Bennett, R. L., J. Wagman, and K. T. Knapp. 1975. The Application of a Multichannel Fixed and Sequential Spectrometer System to the Analysis of Air Pollution Particulate Samples from Source Emissions and Ambient Air. Adv. X-Ray Anal. 19:393-402.

6. Berry, J. W., D. W. Osgood, and P. A. St. John. 1974. Chemical Villains — A Biology of Pollution. The C. V. Mosby Company, St. Louis, Mo. 189 pp.

7. Bondietti, E. A., R. M. Perhac, F. H. Sweeton, and T. Tamura. 1974. Toxic Metals in Sediments. In: Ecology and Analysis of Trace Contaminants, W. Fulkerson, W. D. Shults, and R. I. Van Hook, eds. ORNL/NSF/EATC-6, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 176-194.

8. Bowen, H.J.M. 1966. Trace Elements in Biochemistry. Academic Press, New York. 241 pp.

9. Brady, N. C. 1974. The Nature and Properties of Soils, 8th ed. Macmillan Publishing Company, Inc., New York. 638 pp.

10. Brantley, J. N., J. P. Breillatt, Jr., F. S. Brinkley, C. W. Francis, B. A. Halsall, R. Levy, and S. G. Rush. 1974. Zonal Centrifugation: Applied Aspects in Elucidating Chemical and Biological Forms, Distribution and Availability of Heavy Metals in the Environment. In: Ecology and Analysis of Trace Contaminants, W. Fulkerson, W. D. Shults, and R. I. Van Hook, eds. ORNL/NSF/EATC-6, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 195-221.

11. Burrell, D. C. 1974. Atomic Spectrometric Analysis of Heavy-Metal Pollutants in Water. Ann Arbor Science Publishers, Inc., Ann Arbor, Mich. 331 pp.

12. Camp, D. C., A. L. VanLehn, J. R. Rhodes, and A. H. Pradzynski. 1975. Intercomparison of Trace Element Determinations in Simulated and Real Air Particulate Samples. X-Ray Spectrom. (Great Britain) 4:123-137.

13. Chang, L. W., and J. A. Sprecher. 1976. Pathological Changes in the Kidney after Methyl Cadmium Intoxication. Environ. Res. 12:92-109.

14. Chizhikov, D. M. 1966. Cadmium. Pergamon Press, New York. 263 pp.

15. Coleman, D. M., R. E. Van Atta, and L. N. Klatt. 1972. Device for Field Determination of Selected Heavy Metals in Natural Waters. Environ. Sci. Technol. 6(5):452-455.

16. Emmelin, L. 1973. Cadmium Poisoning from a Soft Drink Machine. Ambio (Norway) 2(1-2):48.

17. Environmental Instrumentation Group. 1973*a*. Instrumentation for Environmental Monitoring — Biomedical. Lawrence Berkeley Laboratory, University of California, Berkeley, Calif.

18. Environmental Instrumentation Group. 1973*b*. Instrumentation for Environmental Monitoring — Water. Lawrence Berkeley Laboratory, University of California, Berkeley, Calif.

19. Environmental Science Associates, Inc., 175 Bedford Street, Burlington, Mass. 01803.

20. Fleischer, M., A. F. Sarofim, D. W. Fassett, P. Hammond, H. T. Shacklette, I.C.T. Nisbet, and S. Epstein. 1974. Environmental Impact of Cadmium: A Review by the Panel on Hazardous Trace Substances. Environ. Health Perspect. 7:253-323.

21. Friberg, L., M. Piscator, and G. Nordberg. 1971. Cadmium in the Environment — I. National Air Pollution Control Administration Publication APTD 0681.

22. Friberg, L., T. Kjellström, G. Nordberg, and M. Piscator. 1975. Cadmium in the Environment — III. EPA-650/2-75-049, U.S. Environmental Protection Agency, Washington, D.C. 218 pp.

23. Friberg, L., M. Piscator, G. F. Nordberg, and T. Kjellström. 1974. Cadmium in the Environment, 2nd ed. CRC Press, Inc., Cleveland, Ohio. 248 pp.

24. Fulkerson, W., and H. E. Goeller, eds. 1973. Cadmium, the Dissipated Element. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. 473 pp.

25. Gamble, D. S., and M. Schnitzer. 1974. The Chemistry of Fulvic Acid and Its Reactions with Metal Ions. In: Trace Metals and Metal-Organic Interactions in Natural Waters. Ann Arbor Science Publishers, Inc., Ann Arbor, Mich. pp. 265-302.

26. Goeller, H. E., E. C. Hise, and H. B. Flora II. 1973. Societal Flow of Zinc and Cadmium. In: Cadmium, the Dissipated Element. W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 61-134.

27. Gross, S. B., E. A. Pfitzer, D. W. Yeager, and R. A. Kehoe. 1975. Lead in Human Tissues. Toxicol. Appl. Pharmacol. 32:638-651.

28. Hahne, H.C.H., and W. Kroontje. 1973. Significance of pH and Chloride Concentration on Behavior of Heavy Metal Pollutants: Mercury(II), Cadmium(II), Zinc(II), and Lead(II). J. Environ. Qual. 2(4):444-450.

29. Harris, R. L., G. R. Helz, and R. L. Cory. 1975. Processess Affecting the Vertical Distribution of Trace Components in the Chesapeake Bay. In: Marine Chemistry in the Coastal Environment, ACS Symposium Series No. 18. American Chemical Society. pp. 176-185.

30. Hem, J. D. 1972. Chemistry and Occurrence of Cadmium and Zinc in Surface Water and Groundwater. Water Resour. Res. 8(3):661-679.

31. Hise, E. C., and W. Fulkerson. 1973. Environmental Impact of Cadmium Flow. In: Cadmium, the Dissipated Element. W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 203-322.

32. International Agency for Research on Cancer. 1973. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man, Vol. 2, Some Inorganic and Organometallic Compounds. Lyon, France. pp. 74-99.

33. Jenne, E. A. 1968. Control on Mn, Fe, Co, Ni, Cu, and Zn Concentrations in Soils and Water: The Significant Role of Hydrous Mn and Fe Oxides. Adv. Chem. Ser. 73:337-387.

34. Kägi, J.H.R., and B. L. Vallee. 1960. Metallothionein: A Cadmium- and Zinc-containing Protein from Equine Renal Cortex. J. Biol. Chem. 235(12):3460-3465.

35. Kägi, J.H.R., and B. L. Vallee. 1961. Metallothionein: A Cadmium and Zinc-containing Protein from Equine Renal Cortex: II. Physicochemical Properties. J. Biol. Chem. 236(9):2435-2442.

36. Kjellström, T., B. Lind, L. Linman, and G. Nordberg. 1974. A Comparative Study on Methods for Cadmium Analysis of Grain with an Application to Pollution Evaluation. Environ. Res. 8:92-106.

37. Kolthoff, I. M., and P. J. Elving. 1961. Treatise on Analytical Chemistry, Part II, Analytical Chemistry of the Elements, Vol. 3. Interscience Publishers, New York. 380 pp.

38. Kometani, T. Y., J. L. Bove, B. Nathanson, S. Siebenberg, and M. Magyar. 1972. Dry Ashing of Airborne Particulate Matter on Paper and Glass Fiber Filters for Trace Metal Analysis by Atomic Absorption Spectrometry. Environ. Sci. Technol. 6(7):617-620.

39. MacLeod, K. E., and R. E. Lee, Jr. 1973. Selected Trace Metal Determination of Spot Tape Samples by Anodic Stripping Voltammetry. Anal. Chem. 45(14):2380-2383.

40. Maeck, W. J., G. L. Booman, M. E. Kussy, and J. E. Rein. 1961. Extraction of the Elements as Quaternary (Propyl, Butyl, and Hexyl) Amine Complexes. Anal. Chem. 33(12):1775-1780.

41. Marx, J. L. 1975. Air Pollution: Effects on Plants. Science 187:731-733.

42. McCaull, J. 1971. Building a Shorter Life. Environment 13(7):3-41.

43. McFarren, E. F., and R. J. Lishka. 1968. Evaluation of Laboratory Methods for the Analysis of Inorganics in Water. Adv. Chem. Ser. 73:253-262.

44. Menden, E. E., V. J. Elia, L. W. Michael, and H. G. Petering. 1972. Distribution of Cadmium and Nickel of Tobacco during Cigarette Smoking. Environ. Sci. Technol. 6(9):830-832.

45. National Academy of Sciences. 1974. Geochemistry and the Environment, Vol. 1, The Relation of Selected Trace Elements to Health and Disease. Washington, D.C. 113 pp.

46. Nordberg, G. F., M. Nordberg, M. Piscator, and O. Vesterberg. 1972. Separation of Two Forms of Rabbit Metallothionein by Isoelectric Focusing. Biochem. J. (Great Britain) 126:491-498.

47. Osteryoung, J. G., and R. A. Osteryoung. 1972. Pulse Polarographic Analysis of Toxic Heavy Metals. Am. Lab. 4(7):8-16.

48. Posselt, H. S. 1971. Environmental Chemistry of Cadmium in Aqueous Systems. Ph.D. Dissertation. University of Michigan, Ann Arbor, Mich. 189 pp.

49. Posselt, H. S., and W. J. Weber, Jr. 1971. Environmental Chemistry of Cadmium in Aqueous Systems. University of Michigan, Ann Arbor, Mich. 178 pp.

50. Posselt, H. S., and W. J. Weber, Jr. 1974. Studies on the Aqueous Corrosion Chemistry of Cadmium. In: Aqueous-Environmental Chemistry of Metals, A. J. Rubin, ed. Ann Arbor Science Publishers Inc., Ann Arbor, Mich. pp. 291-315.

51. Pourbaix, M. 1973. Lectures on Electrochemical Corrosion. Plenum Press, New York. 336 pp.

52. Ross, R. T., and J. G. Gonzalez. 1974. The Direct Determination of Cadmium in Biological Samples by Selective Volatilization and Graphite Tube Reservoir Atomic Absorption Spectrometry. Anal. Chim. Acta (Netherlands) 70:443-447.

53. Sandell, E. B. 1959. Colorimetric Determination of Traces of Metals, 3rd ed. Interscience Publishers, Inc., New York. 1032 pp.

54. Shacklette, H. T. 1972. Cadmium in Plants. U.S. Geological Survey Bulletin 1314-G, U.S. Government Printing Office, Washington, D.C. 28 pp.

55. Sheverdina, N. I., and K. A. Kocheshkov. 1967. The Organic Compounds of Zinc and Cadmium, Vol. 3 of Series: Methods of Elemento-Organic Chemistry, A. M. Nesmeyanov and K. A. Kocheskov, eds. North-Holland Publishing Company, Amsterdam, Netherlands. 252 pp.

56. Shults, W. D., W. S. Lyon, and J. A. Carter. 1973. The Analytical Determination of Cadmium in the Environment. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 427-442.

57. Torchinskii, Y. M. 1974. Sulfhydryl and Disulfide Groups of Proteins. Consultants Bureau, New York. 275 pp.

58. U.S. Environmental Protection Agency. 1972. Handbook for Analytical Quality Control in Water and Wastewater Laboratories. Cincinnati, Ohio.

59. U.S. Environmental Protection Agency. 1974. Methods for Chemical Analysis of Water and Wastes. EPA-625/6-74-003, Washington, D.C. 298 pp.

60. Van Hook, R. I., et al. 1973. Toxic Materials in the Environment. In: Environmental Sciences Division Annual Progress Report, Period Ending September 30, 1973. ORNL-4848, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 52-58.

61. von Lehmden, D. J., R. H. Jungers, and R. E. Lee, Jr. 1974. Determination of Trace Elements in Coal, Fly Ash, Fuel Oil, and Gasoline — A Preliminary Comparison of Selected Analytical Techniques. Anal. Chem. 46(2):239-244.

62. Watanabe, H., S. Berman, and D. S. Russell. 1972. Determination of Trace Metals in Water Using X-ray Fluorescence Spectrometry. Talanta (Great Britain) 19(11):1363-1375.

63. Watson, M. R. 1973. Cadmium Removal from Water. In: Pollution Control in Metal Finishing. Noyes Data Corporation, Park Ridge, N.J. pp. 87-89.

64. Weast, R. C., ed. 1971. Handbook of Chemsitry and Physics, 51st ed. The Chemical Rubber Company, Cleveland, Ohio.

65. Weiss, H. V., K. Chew, M. Guttman, and A. Host. 1974. The Determination of Cadmium in Sea Water by Radioactivation. Anal. Chim. Acta (Netherlands) 73:173-176.

66. Wood, J. M. 1974. Biological Cycles for Toxic Elements in the Environment. Science 183:1049-1052.

67. Yeager, D. W., J. Cholak, and B. G. Meiners. 1973. Determination of Cadmium in Biological and Related Materials by Atomic Absorption. Am. Ind. Hyg. Assoc. J. 34:450-454.

68. Zirino, A., and M. L. Healy. 1972. pH-Controlled Differential Voltammetry of Certain Trace Transition Elements in Natural Waters. Environ. Sci. Technol. 6(3):243-249.

SECTION 3

BIOLOGICAL ASPECTS IN MICROORGANISMS

3.1 SUMMARY

Zooplankton and phytoplankton can absorb and concentrate trace metals present in water. Cadmium can be found adsorbed to exoskeletons of zooplankton in bottom sediments. It is concentrated by several species of algae, some of which tolerate high concentrations of cadmium in the laboratory. Because cadmium is present in water, its effects on microorganisms must be elucidated in order to permit evaluation of hazards as the metal enters the food chain.

In a small number of studies, cadmium was found to be toxic in varying concentrations to several species of bacteria, yeast, and fungi. In general, cadmium levels needed to cause toxic effects in bacteria (0.2 to 6 ppm) are several times higher than concentrations generally detected in waters polluted with industrial waste. The small amount of available data concerning yeast and fungi does not permit a comparison of toxic levels with concentrations in the environment. Anaerobic digestion of sludge is inhibited by soluble cadmium, as influenced by pH, and by the concentrations of sulfide and carbonate ions. Several fungicides contain cadmium compounds as the active ingredient, but little is known about mode of action or lethal concentrations.

3.2 METABOLISM

3.2.1 Bacteria

Information available on the relationship of cadmium to bacterial metabolism is limited. Mitchell (1974) stated that metabolism of cadmium by bacteria has not been detected in natural habitats. Kondo, Ishikawa, and Nakahara (1974) reported that *Staphylococcus aureus*, a common human pathogen, contains a specific gene in the penicillinase plasmid that confers resistance to cadmium toxicity. Experimentally, a 2-ppb concentration of cadmium chloride proved toxic to the plasmid-negative strain, while a concentration of 200 ppb was needed before a toxic effect on the plasmid-positive strain was seen. This resistance was attributed to some mechanism which retained the cadmium ion outside the cell by conformational changes of the cytoplasmic membrane and/or to a recognition mechanism allowing the cell to distinguish between toxic cadmium ions and beneficial calcium ions. The bacteria not carrying the plasmid incorporated cadmium ions into the cell. This incorporation was temperature dependent and did not occur at 4°C. Kondo, Ishikawa, and Nakahara (1974) concluded that cadmium ions could not penetrate resting cells of *S. aureus* and suggested that cadmium ions may uncouple oxidative phosphorylation in the cytoplasmic membranes. Chopra (1971) reported earlier that most of the cadmium ions taken up by the cadmium-sensitive cells were bound to a structure within the cell and were not merely adsorbed to the surface.

Cadmium increased the swelling of *Pseudomonas aeruginosa* cells which had been pretreated with alkali (Bernheim, 1973). These treated cells also showed an increased efflux of potassium in the presence of cadmium. Bernheim postulated that cadmium can react with membrane phospholipids since it precipitates phospholipids from solution.

3.2.2 Yeast

Some research has been done on effects of cadmium on respiration in yeasts. Cadmium ions at a concentration of 1.5 to 150 μM (0.17 to 17 ppm) stimulated the oxidation of reduced nicotinamide adenine dinucleotide (NADH) by *Saccharomyces cerevisiae* mitochondria (Subik and Kolarov, 1970). The cadmium-stimulated respiratory rate exceeded state 3 oxidation of NADH but was sensitive to antimycin A inhibition. Even though stimulation was found over a wide range of concentrations, greatest stimulation occurred between 5 and 15 μM. Cadmium inhibited citrate oxidation to a lesser extent than succinate oxidation (Subik and Kolarov, 1970). The authors suggested that the effect of cadmium involves reaction with SH groups and is associated with an increased cation permeability of the mitochondrial inner membrane, previously reported by Blondin, Vail, and Green (1969). Cells of bakers' yeast, grown in broth containing 10 ppm cadmium, showed a conspicuous endoplasmic reticulum in electron micrographs (Lindegren and Lindegren, 1973) in contrast to cells grown in absence of the metal. Only a few mitochondria with signs of cristae were observed, and no normal cristate mitochondria were seen. Cells grown in cadmium broth were replated on normal agar; 30% of the colonies formed were "petite" and respiratory deficient. The authors concluded that the cadmium ion produced a nonlethal modification of the mitochondria, which caused them to lose respiratory capacity.

3.2.3 Plankton

Only scattered data are available concerning the effects of cadmium on plankton. Abdullah, Royle, and Morris (1972) suggested that phytoplankton and zooplankton are natural agencies for removal and control of trace metals. When the concentration of trace metals increased, a corresponding decrease of plankton took place, suggesting a concentration-effect relationship. Knauer and Martin (1973) also reported an inverse relationship between cadmium concentration and phytoplankton productivity. This connection, however, was obscured by changes in cadmium concentration caused by ocean swells. Studies by Rice, Leighty, and McLeod (1973) suggest that, for the most part, accumulation of trace metals by plankton is due to surface adsorption and ion exchange processes.

3.3 EFFECTS

Cadmium toxicity to microorganisms is summarized in Table 3.1.

3.3.1 Bacteria

Knowledge concerning the effects of cadmium on bacteria is limited. The few reported studies determined the sensitivity of the test organism

TABLE 3.1. CADMIUM CONCENTRATIONS CAUSING TOXICITY TO VARIOUS MICROORGANISMS

Organism	Toxic concentration	Reference
Escherichia coli	6 ppm Cd as $CdCl_2$[a]	Zwarun, 1973
Staphylococcus aureus		Kondo, Ishikawa, and Nakahara, 1974
Plasmid-positive	0.2 ppm $CdCl_2$	
Plasmid-negative	2 ppb $CdCl_2$	
Selenastrum capricornutum	0.65 ppm Cd[b]	Barlett, Rabe, and Funk, 1974
Chaetoceros galvestonensis	0.1 ppm (not toxic)	Hannan and Patouillet, 1972
Cyclotella nana	0.1 ppm (not toxic)	Hannan and Patouillet, 1972
Phaeodactylum tricornutum	0.1 ppm (not toxic)	Hannan and Patouillet, 1972
Tetrahymena pyriformis	1.67 ppm $CdSO_4$[a]	Carter and Cameron, 1973
Thraustochytrium striatum	0.112 ppm[c] 1.12 ppm as $CdCl_2$[d]	Schneider, 1972 Schneider, 1972

[a] Lethal threshold concentration.
[b] Algicidal concentration.
[c] Suppressed zoospore activity.
[d] Suppressed sporangia activity.

to cadmium and, for the most part, did not delve into the mode of action responsible for the toxic effects. As in the case of the interaction between zinc and cadmium in higher animals, the toxic action in bacteria may also involve zinc enzymes such as carboxypeptidase (Zwarun, 1973).

The effects of various concentrations of cadmium on *Escherichia coli* were investigated by measuring $^{14}CO_2$ production using ^{14}C-glucose as substrate (Figure 3.1) (Zwarun, 1973); the organism exhibited a high tolerance for cadmium. At concentrations below 0.6 ppm cadmium as $CdCl_2$, no effect on $^{14}CO_2$ production was observed. However, with a tenfold increase in cadmium concentration to 6 ppm, an inhibitory threshold level was reached and $^{14}CO_2$ production began to decrease. The decrease in CO_2 evolution could be correlated with a reduction in the number of surviving cells. Zwarun (1973) suggested that cadmium might be associated with the cell wall and outer membrane and that only a small percentage of cadmium taken up actually penetrates into the cytoplasm.

Studies have shown that cadmium interferes with anaerobic digestion of sludge. Mosey (1971), for example, found cadmium inhibition to be both quick acting and only partially reversible. Inhibition was a function of contact time and shock-dose concentration. A contact time of only 5 min produced inhibition, and concentrations of 50 to 300 ppm proved toxic. A shock-dose concentration was more toxic than gradual accumulation to the same concentration. Mosey (1971) proposed that in the case of the gradual accumulation group, slow precipitation of the metal occurred until the

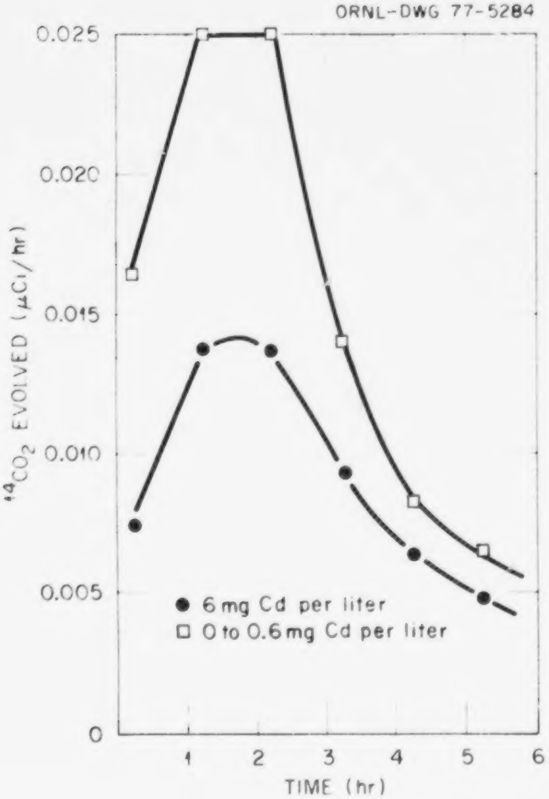


Figure 3.1. Effect of cadmium concentration on $^{14}CO_2$ evolution by *Escherichia coli* (10^8 cells). Source: Adapted from Zwarun, 1973, Figure 2, p. 355.

neutralizing capacity of the sludge had been exhausted. Sulfide was found to obviate the cadmium effect with an optimum molar ratio for neutralization of 1.0. The resultant product from this reaction was insoluble cadmium sulfide. Cadmium toxicity to anaerobic microorganisms depends on pH above 7.0; at higher pH values, insoluble cadmium carbonate is formed. Mosey, Swanwick, and Hughes (1971) reported that the neutralization of cadmium toxicity depends on the total soluble sulfide rather than the free sulfide ion concentration.

3.3.2 Algae

Cadmium produces algicidal and algistatic effects on the freshwater unicellular green alga *Selenastrum capricornutum*. Growth rate begins to decrease at a concentration of 50 ppb; higher concentrations both depress growth rate and prolong the lag phase (Figure 3.2). At 650 ppb cadmium was algicidal (Bartlett, Rabe, and Funk, 1974). Earlier studies by Hannan and Patouillet (1972), however, involving the marine algae *Phaeodactylum tricornutum*, *Cyclotella nana*, and *Chaetoceros galvastonensis* demonstrated that cadmium at a concentration of 0.1 ppm is not toxic. Species specificity may be responsible for such differences.

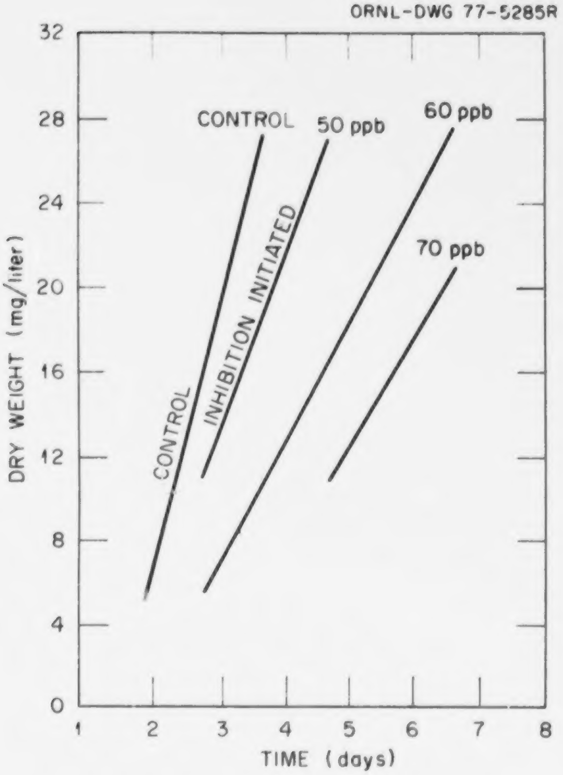


Figure 3.2. Growth rate of *Selenastrum capricornutum* as a function of cadmium concentration in culture medium. Source: Adapted from Bartlett et al., 1974, Figure 3, p. 182. Reprinted by permission of the publisher.

3.3.3 Protozoa

Even though protozoa occupy an important part of the food chain, the effects of heavy metals have been ignored. Carter and Cameron (1973) developed an assay using *Tetrahymena pyriformis*, a filter-feeding ciliated protozoan, as the test organism for heavy-metal toxicity. Of the metals tested — cadmium, cobalt, lead, mercury, and zinc — cadmium was the most toxic. Concentrations of cadmium sulfate from 0.84 to 3.33 ppm killed at least 20% of the cells within 30 min; 3.33 to 10.00 ppm killed all cells. The lethal threshold was 1.67 ppm.

Because a rapid and reliable bioassay is needed for the determination of harmful effects of trace metals in water, such single cell techniques may prove very useful. It is worth noting that cell cultures from higher animals have also been used for this purpose (Christian et al., 1973).

3.3.4 Fungi

Cadmium has been used in various fungicides, especially in turf grass fungicides and sprays for fruit trees. The nature of the cadmium effect is not known.

A study to determine the adequacy of using the water mold *Thraustochytrium striatum* as a test organism for pollutants in seawater was carried out by Schneider (1972). A concentration of 0.112 ppm cadmium completely suppressed zoospore activity, and only below 11 ppb cadmium could normal zoospore activity be found. At a concentration of 1.12 ppm cadmium, some sporangia were observed and some were still viable when subcultured.

SECTION 3

REFERENCES

1. Abdullah, M. I., L. G. Royle, and A. W. Morris. 1972. Heavy Metal Concentration in Coastal Waters. Nature (Great Britain) 235:158-160.

2. Bartlett, L., F. W. Rabe, and W. H. Funk. 1974. Effects of Copper, Zinc and Cadmium on *Selenastrum capricornutum*. Water Res. (Great Britain) 8:179-185.

3. Bernheim, F. 1973. Effect of Na Salts of Organic Acids on Acid Induced Swelling and K Loss in *Pseudomonas aeruginosa* and Effect of pH on Actions of Mercuric, Cadmium, and Zinc Salts. Cytobios (Great Britain) 8:7-13.

4. Blondin, G. A., W. J. Vail, and D. E. Green. 1969. The Mechanism of Mitochondrial Swelling: II. Pseudoenergized Swelling in the Presence of Alkali Metal Salts. Arch. Biochem. Biophys. 129:158-172.

5. Carter, J. W., and I. L. Cameron. 1973. Toxicity Bioassay of Heavy Metals in Water Using *Tetrahymena pyriformis*. Water Res. (Great Britain) 7:951-961.

6. Chopra, I. 1971. Decreased Uptake of Cadmium by a Resistant Strain of *Staphylococcus aureus*. J. Gen. Microbiol. (Great Britain) 63:265-267.

7. Christian, R. T., T. E. Cody, C. S. Clark, R. Lingg, and E. J. Cleary. 1973. Development of a Biological Chemical Test for the Potability of Water. Am. Inst. Chem. Eng. Symp. Ser. 70:15-21.

8. Hannan, P. J., and C. Patouillet. 1972. Effect of Mercury on Algal Growth Rates. Biotechnol. Bioeng. (Great Britain) 14:93-101.

9. Knauer, G. A., and J. H. Martin. 1973. Seasonal Variations of Cadmium, Copper, Manganese, Lead, and Zinc in Water and Phytoplankton in Monterey Bay, California. Limnol. Oceanogr. 18(4):597-604.

10. Kondo, I., T. Ishikawa, and H. Nakahara. 1974. Mercury and Cadmium Resistance Mediated by the Penicillinase Plasmid in *Staphylococcus aureus*. J. Bacteriol. 117(1):1-7.

11. Lindegren, C. C., and G. Lindegren. 1973. Mitochondrial Modification and Respiratory Deficiency in the Yeast Cell Caused by Cadmium Poisoning. Mutat. Res. (Netherlands) 21:315-322.

12. Mitchell, R. 1974. Metals as Pollutants. In: Introduction to Environmental Microbiology. Prentice-Hall, Inc., Englewood Cliffs, N.J. p. 226.

13. Mosey, F. E. 1971. The Toxicity of Cadmium to Anaerobic Digestion: Its Modification by Inorganic Anions. Water Pollut. Control (Great Britain) 70:584-598.

14. Mosey, F. E., J. D. Swanwick, and D. A. Hughes. 1971. Factors Affecting the Availability of Heavy Metals to Inhibit Anaerobic Digestion. Water Pollut. Control (Great Britain) 6:2-12.

15. Rice, H. V., D. A. Leighty, and G. C. McLeod. 1973. The Effects of Some Trace Metals on Marine Phytoplankton. In: Crit. Rev. Microbiol. 3:27-49.

16. Schneider, J. 1972. Niedere Pfilze als Testorganismen für Schadstoffe in Meer- und Brackwasser Die Wirkung von Schwermetallverbindungen und Phenol auf *Thraustochytrium striatum* (Lower Fungi as Test Organisms of Pollutants in Sea and Brackish Water: The Effects of Heavy Metal Compounds and Phenol on *Thraustochytrium striatum*). Mar. Biol. (West Germany) 16:214-225.

17. Subik, J., and J. Kolarov. 1970. Metabolism of Cadmium and Effects of Divalent Cations on Respiratory Activity of Yeast Mitochondria. Folia Microbiol. (Czechoslovakia) 15(6):448-458.

18. Zwarun, A. A. 1973. Tolerance of *Escherichia coli* to Cadmium. J. Environ. Qual. 2(3):353-355.

SECTION 4

BIOLOGICAL ASPECTS IN PLANTS

4.1 SUMMARY

Background concentrations of cadmium in plants appear to be less than 1 ppm. Generally, plants accumulate this element when environmental background levels are exceeded. Cadmium is readily transported to all regions of the plant and often accumulates in the roots. No natural means for elimination of cadmium from living plant tissue is known.

Cadmium concentrations which reduce plant yields are variable and largely unknown. Cadmium interferes with stomatal function, chlorophyll production, ion absorption and desorption, and photosynthesis.

4.2 METABOLIC PROCESSES

The concentration of cadmium in plants is determined by the ability of the plant species to take up the metal and by the cadmium concentration in the environment. Plant cadmium content usually reflects environmental cadmium content. When the cadmium content of soil is higher than background levels, the cadmium content of plant tissue tends to be increased (Table 4.1).

4.2.1 Uptake and Absorption

Studies on plants grown in soil or nutrient solution indicate that cadmium may be absorbed by either roots or foliage and incorporated into plant tissue. Although conditions regulating this uptake are not well understood, several determining factors are known and are discussed in this section. In general, it can be stated that cadmium is accumulated by a wide variety of plant species to an extent dependent upon the cadmium concentration of the substrate (Bingham et al., 1975), its pH, and other factors. For example, John (1972) applied lime to soil supporting radish plants (*Raphanus sativus*) and found a decrease in cadmium uptake (Figure 4.1). The pH of unlimed soil was 4.1; after application of lime the pH rose to 5.5. Cadmium uptake by both tops and roots was significantly higher from unlimed (low pH) soil.

Similar results have been obtained for a variety of plants (John, VanLaerhoven, and Chuah, 1972; Lagerwerff, 1971; Lagerwerff and Specht, 1971; Linnman et al., 1973; Williams and David, 1973). Francis and Rush (1973) evaluated the availability of cadmium chloride (water soluble) and cadmium oxide (water insoluble) to Japanese millet (*Echinochloa frumentacea*) at three liming levels (Table 4.2). After 26 days, cadmium concentrations in plants grown in presence of $CdCl_2$ were two to three times higher than in those exposed to CdO; the availability of cadmium in both cases was greatest at low pH. A second harvest after an additional 63 days revealed no significant difference in cadmium content from either form in limed treatments but greater concentrations from $CdCl_2$ in unlimed treatments. Francis and Rush (1973) suggested that the cadmium originally applied became less

TABLE 4.1. ESTIMATES OF CADMIUM CONCENTRATIONS IN SOME PLANT TISSUES

Plant or plant part	Reported or estimated cadmium concentration (ppm, dry wt)	
	In environments presumably having normal cadmium levels	In environments having greater than normal cadmium levels
Marine algae	0.1-1	
Mosses (bryophytes)	0.7-1.2	8-340
Lichens (fruticose type)	0.1-0.4	1
Grasses	0.03-0.3	0.6-40
Grains		
Corn (*Zea mays*)	0.1	2
Rice (polished)		0.5
Barley, wheat, and oats	0.1-0.5	0.1-1.5[a]
Vegetables		
Asparagus		8
Beet root	0.05	0.24
Cabbage leaves	0.05	6-12
Carrots	<0.35	8
Chinese cabbage		41
Eggplant fruit		8
Kale	1	
Leafy vegetables used as pot herbs or salads	0.3-0.5	3-50
Leeks		14
Lettuce	0.3-0.5	4-16
Potatoes	0.05-0.3	0.6-2
Spinach	0.6-1.2	
Turnip roots		5
Turnip leaves		15
Tomatoes		2
Trees, deciduous		
Leaves	0.1-2.4	4-17
Stems (branches)	0.1-1.3	0.14-1.5
Trees, coniferous		
Leaves	0.1-0.9	0.05-1
Stems		0.03-1.5
Epiphytes (Spanish moss)	0.1	1
Floating aquatic plants (duckweed)		17
Marine flowering plants (*Zostera marina*)	0.23	

[a] Original data given in wet weight; because the water content of grains is very low, these values were not converted to a dry weight basis.

Source: Adapted from Shacklette, 1972, Table 3, p. 24.

ORNL-DWG 77-5256

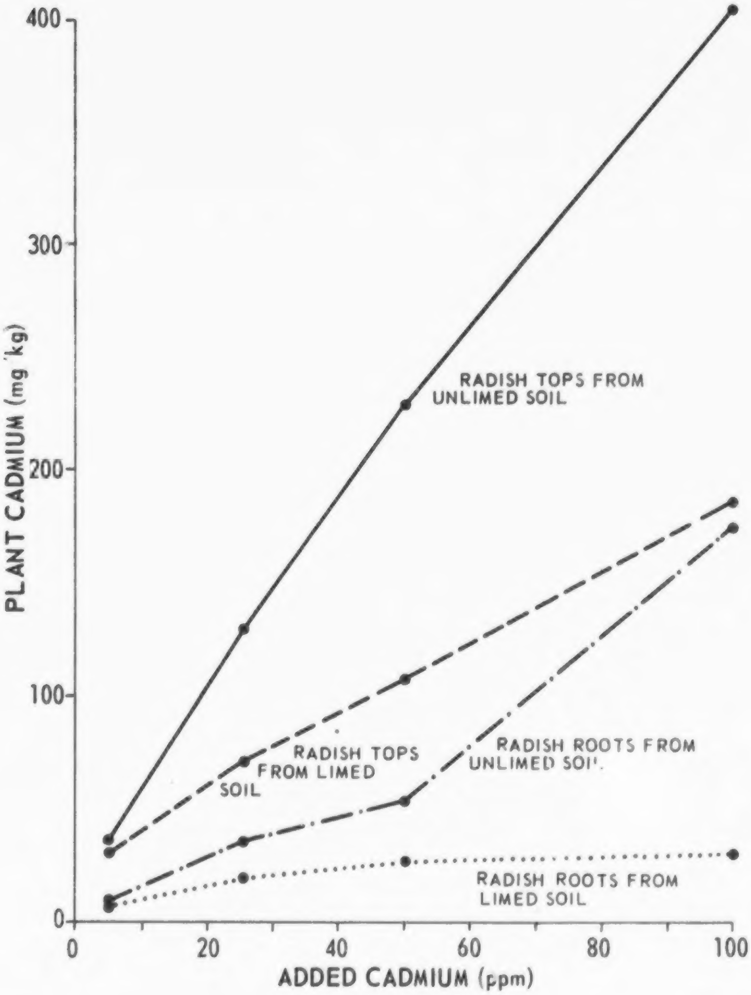


Figure 4.1. Mean cadmium concentrations in radish tops and roots grown on unlimed (pH 4.1) and limed (pH 5.5) soils containing various concentrations of added cadmium. Source: John, 1972, Figure 1, p. 717. Reprinted by permission of the publisher.

available during the second crop due to replacement of Ca^{2+} by Cd^{2+} in the $CaCO_3$ crystals and/or a stronger chelation of soil cadmium at the higher pH. In contrast to experimental studies conducted in soil, studies of bush beans (*Phaseolus vulgaris*) grown in nutrient solutions revealed no significant difference in cadmium uptake between pH 4.6 and pH 6.5. However, when the iron chelate of diethylenetriamine pentaacetic acid was present in the nutrient solution, greater cadmium uptake occurred at pH 4.6 than at pH 6.5 (Francis and Rush, 1973); this fact presumably reflects greater chelation of cadmium at the higher pH.

TABLE 4.2. CONCENTRATIONS OF ^{109}Cd IN JAPANESE MILLET DERIVED FROM TWO CHEMICAL FORMS OF CADMIUM AT THREE LEVELS OF CALCIUM CARBONATE[a]

$CaCO_3$ level (tons/ha)	^{109}Cd concentration (pCi/mg dry wt)		
	First harvest, total plant	Leaves	Heads
	Derived from $CdCl_2$		
0	46.4 *a*	14.9 *a*	4.6 *a*
11	45.4 *a*	3.6 *bc*	0.79 *c*
22	29.5 *b*	4.0 *bc*	0.46 *c*
	Derived from CdO		
0	26.3 *bc*	6.8 *b*	2.4 *b*
11	15.7 *d*	2.6 *c*	0.62 *c*
22	18.1 *cd*	2.2 *c*	0.56 *c*

[a] Average of three replicates. Numbers followed by identical letters within the same column are not statistically different at the 5% level as tested by Duncan's multiple range test. Concentration of cadmium in soil <20 ppb; specific activity 1.91 Ci/g.

Source: Adapted from Francis and Rush, 1973, Table V. Reprinted by permission of the publisher.

The influence of zinc on cadmium uptake has also been studied. Haghiri (1974) found that addition of 5 to 50 ppm zinc to soil significantly increased the cadmium concentration of soybean (*Glycine max*) shoots; conversely, at 100 ppm, zinc reduced cadmium uptake. Lagerwerff and Biersdorf (1972) used complete nutrient solutions with 2, 20, or 100 ppb cadmium combined with 20, 100, or 400 ppb zinc to determine whether zinc affects cadmium uptake in radishes. In addition, plants grown in a greenhouse at 100% relative humidity (to decrease transpiration) were compared with those grown outdoors near a highway. In both cases, cadmium uptake at 2 and 20 ppb cadmium decreased with increasing zinc content, while at the 100 ppb cadmium concentration higher zinc concentrations resulted in increased cadmium uptake. This was thought to be the result of root damage because growth was also decreased at high cadmium concentrations. Because high zinc concentrations are also detrimental to growth, this study was criticized by Smith and Huckabee (1973) for lacking a control of zinc without cadmium. Subsequently, Van Hook et al. (1974) concluded that increased cadmium uptake by plants in the presence of zinc cannot be attributed simply to root damage. Significant correlations between concentrations of cadmium and of a variety of other elements, including zinc, were found under various conditions in tops and roots of radish and lettuce (John, VanLaerhoven, and Chuah, 1972). These results are collected in Table 4.3.

TABLE 4.3. CORRELATION COEFFICIENTS BETWEEN LEVELS OF CADMIUM AND LEVELS OF OTHER ELEMENTS IN THE SAME PLANT PART

Factor correlated	Zinc	Nickel	Copper	Iron	Magnesium	Calcium	Phosphorus	Sulfur
Cadmium in radish tops	0.15	0.77[a]	0.56[a]	0.73[a]	0.06	0.14	0.14	0.01
Cadmium in radish roots	0.79[a]	0.96[a]	0.74[a]	0.79[a]	0.86[a]	0.81[a]	0.25	0.13
Cadmium in lettuce tops	0.49[a]	0.58[a]	0.37[b]	0.20	0.10	0.37	0.04	0.45
Cadmium in lettuce roots	0.22	0.52[a]	0.28	0.09	0.12	0.25	0.16	0.26

[a] Significant at 1% level.
[b] Significant at 5% level.

Source: Adapted from John, VanLaerhoven, and Chuah, Table III, p. 1006. Reprinted by permission of the publisher.

Hutchinson and Czyrska (1972) found that zinc concentrations up to 0.08 ppm in solution increased cadmium uptake by aquatic plants. Francis and Rush (1973) also found enhanced cadmium uptake by plants in the presence of zinc. Nutrient solutions containing 5 ppm zinc resulted in higher plant cadmium concentrations than did solutions containing 1 ppm zinc. Zinc increased cadmium concentrations in stem and leaf tissues but decreased that in roots. Nitrogen added as ammonium nitrate significantly lowered net tracer uptake by foliage but not by root or stem. Significantly higher cadmium concentrations appeared in the upper leaves at all nitrogen concentrations employed, suggesting that cadmium translocation occurred early in leaf development. In these experiments, selenium was also tested; concentrations of 0.1 ppm severely reduced foliar cadmium concentrations.

Investigations of cadmium-zinc interactions also include studies on effects of high cadmium concentrations on zinc accumulation. Bingham et al. (1975) determined that the zinc concentration of plant tissues was significantly reduced at higher cadmium treatment rates. Various species of plants were grown to harvest stage on soil pretreated with municipal sewage sludge (1%) moistened with variable amounts of cadmium sulfate. For field beans, the leaf zinc dropped from 47 μg/g for control plants to 24 μg/g for plants grown in soil treated with 160 μg of cadmium as cadmium sulfate per gram of soil. Seed zinc concentrations also decreased in these field bean plants. However, the plants showed no signs of zinc deficiency. Bingham et al. (1975) were unable to offer an adequate explanation for the reduced zinc levels in plants grown in presence of high cadmium sulfate levels.

A survey of the cadmium content in wheat led Huffman and Hodgson (1973) to conclude that superphosphate fertilizers do not affect plant cadmium levels. No significant differences were found between cadmium levels in plants of the eastern United States, an area of heavy superphosphate usage, and in those of the Midwest, an area of little usage.

More recently Reuss, Dooley, and Griffis (1976) tested cadmium uptake by radish, lettuce, and peas from phosphate fertilizer applied over a range equivalent to 0 to 0.087 μg cadmium per gram of soil. These cadmium concentrations represent those commonly found in phosphate fertilizers. Plants grown on a calcareous silt loam soil (pH 8.4) accumulated marginally detectable amounts of cadmium, even at the highest level tested. However, plants grown on a coarse-textured acid soil (pH 4.5) reached cadmium levels of 3.55, 6.01, and 0.53 ppm in radish roots, lettuce tops, and pea seeds respectively. These investigators cautioned that although "the biological significance to consumer organisms is not clear, . . . it does not appear that it can be dismissed as unimportant."

Williams and David (1973) analyzed vegetation from adjacent fertilized and unfertilized fields (Table 4.4). A top-dressing of superphosphate resulted in increased cadmium content of grasses and clover. Controlled studies with superphosphate showed that the cadmium content of oats was 0.02 ppm on unfertilized soil and 0.28 ppm on fertilized soil. Corresponding concentration ranges for subterranean clover and lucerne were 0.10 to 1.34 ppm and 0.03 to 0.60 ppm respectively. The cadmium content of 21 Australian phosphatic fertilizers analyzed by Williams and David (1973) ranged between 18

TABLE 4.4. CADMIUM CONTENT OF PASTURE SPECIES FROM ADJACENT FERTILIZED AND UNFERTILIZED PASTURES

Unfertilized pasture		Fertilized pasture		
Species	Cadmium content (ppm)	Total superphosphate applied (kg/ha)	Species	Cadmium content (ppm)
Native grasses *(Danthonia, Themeda)*	0.028	2500	*Phalaris*	0.060
		2500	Subterranean clover	0.324
Phalaris	0.020	2060	*Phalaris*	0.072
Subterranean clover	0.048	2060	Subterranean clover	0.164
		2800	Ryegrass	0.153
		2800	Subterranean clover	0.411

Source: Adapted from Williams and David, 1973, Table 11, p. 53. Reprinted by permission of the publisher.

and 91 ppm. Swedish fertilizers have been shown to contain 0.1 to 28 ppm cadmium (Stenström and Vahter, 1974), while American brands ranged from 3.48 to 14.3 ppm (Yost et al., 1973) and from 7.4 to 156 ppm cadmium (Reuss, Dooley, and Griffis, 1976).

Oats cultured by John, Chuah, and VanLaerhoven (1972) in contaminated (46.4 ppm cadmium) and uncontaminated (1.3 ppm) soil contained 16.1 and 0.51 ppm cadmium respectively. John (1973) measured cadmium in plants grown on soil containing either 0, 40, or 200 mg cadmium per 1000 g soil. As soil cadmium increased, uptake by oats increased. The highest accumulation occurred in roots, the lowest in grain (Table 4.5).

Williams and David (1973) examined the distribution of cadmium in cereal plants from contaminated and uncontaminated soils (Table 4.6). Five to eighteen percent of the cadmium in plant tops was in the grain. Less than 0.2% of the added cadmium was found in the grain of wheat, oats, and Hungarian rice and about 1% was found in that of Taichung rice. The harvested oat grain included the glumes, which analysis from one sample revealed to contain 25% of the "grain" cadmium.

Uptake of cadmium by barley (*Hordeum vulgare*) from nutrient solutions (cadmium content, 1 ppm) was studied by Cutler and Rains (1974). The cadmium content of both roots and shoots increased over a 28-day growth period. Roots accumulated 250 ppm and shoots 56 ppm. Page, Bingham, and Nelson (1972) found increased uptake by barley and other plants growing in solution culture when the cadmium concentration was raised over the range of 0.1 to 10 ppm. Plants varied in their tolerance to cadmium, but in all species studied the leaves accumulated the metal to much higher concentrations than that present in the culture solution.

Traynor and Knezek (1973) tested the response of corn (*Zea mays*) to contaminated soil. As cadmium increased from 0 to 562 ppm, cadmium content

TABLE 4.5. INFLUENCE OF CADMIUM CONTAMINATION OF SOIL ON CADMIUM CONTENT OF VARIOUS PARTS OF EIGHT CROPS

Crop	Variety	Plant part	Plant cadmium content (ppm)[a]		
			Control	40 mg Cd per 1000 g soil	200 mg Cd per 1000 g soil
Leafy vegetables					
Leaf lettuce, *Lactuca sativa* L., cv. *crispa*	Salad Bowl	Leaves	4.8 *a*	51.1 *a*	667.7 *b*
		Roots	2.3 *a*	294.7 *a*	1628.2 *b*
Spinach, *Spinacia oleracea* L.	King of Denmark	Leaves	12.2 *a*	207.5 *b*	239.3 *b*
		Roots	8.5 *a*	213.9 *b*	490.5 *c*
Brassicas					
Broccoli, *Brassica oleracea* L., cv. *italica*	Waltham No. 29	Leaves	2.7 *a*	36.0 *a*	268.5 *a*
		Roots	6.5 *a*	153.0 *a*	1647.4 *b*
Cauliflower, *Brassica oleracea* L., cv. *botrytis*	Snowball Y	Leaves	4.8 *a*	18.5 *a*	198.6 *b*
		Roots	1.8 *a*	202.5 *a*	1356.7 *b*
Legume					
Peas, *Pisum sativum* L.	Dark Skin Perfection	Seeds	5.4 *a*	101.1 *b*	19.7 *c*
		Pods	5.7 *a*	9.5 *a*	28.2 *b*
		Vines	7.4 *a*	37.2 *a*	116.9 *b*
		Roots	8.6 *a*	409.7 *b*	1571.2 *c*
Grain					
Oats, *Avena sativa* L.	Fraser	Grains	3.9 *a*	20.8 *b*	33.6 *c*
		Husks	4.5 *a*	37.8 *b*	96.0 *c*
		Leaves	3.9 *a*	45.4 *b*	177.0 *c*
		Stalks	3.9 *a*	47.4 *b*	116.5 *c*
		Roots	3.6 *a*	144.8 *a*	663.2 *b*
Tuberous vegetables					
Radish, *Raphanus sativus* L.	Early Scarlet Globe	Tops	9.8 *a*	264.7 *b*	398.0 *b*
		Tubers	3.6 *a*	54.6 *b*	123.3 *c*
Carrot, *Daucus carota* L., cv. *sativa*	Chatenay Red Cored	Tops	6.6 *a*	79.3 *b*	294.4 *c*
		Tubers	2.4 *a*	26.8 *b*	29.8 [illegible]
		Roots	8.5 *a*	1737.5 *b*	6121.5 *c*

[a] Values followed by the same letter do not differ significantly at the 5% level for a given plant part.

Source: Adapted from John, 1973, Table 1, p. 10. Reprinted by permission of the publisher.

TABLE 4.6. DISTRIBUTION OF CADMIUM IN CEREAL PLANTS GROWN IN POT CULTURE

Species	Treatment	Roots		Straw		Grain		Grain cadmium, as percent of cadmium in plant tops
		Content (ppm Cd)	Amount (µg Cd)	Content (ppm Cd)	Amount (µg Cd)	Content (ppm Cd)	Amount (µg Cd)	
Oats (Avon)	Nil	0.08	0.68	0.04	0.37	0.02	0.08	17.8
	200 µg Cd	0.50	2.68	0.24	1.92	0.04	0.25	11.5
Wheat (Robin)	Nil	0.10	0.38	0.06	0.27	0.01	0.04	12.9
	200 µg Cd	0.34	1.30	0.43	1.60	0.11	0.35	17.9
Rice (Hungarian)	Nil	0.19	1.64	0.05	0.45	0.01	0.07	13.2
	200 µg Cd	4.00	29.30	0.86	7.76	0.05	0.43	5.2
Rice (Taichung)	Nil	0.27	4.66	0.10	2.12	0.02	0.30	12.4
	200 µg Cd	1.27	22.10	0.53	10.50	0.14	2.20	17.3

Source: Adapted from Williams and David, 1973, Table 9, p. 52. Reprinted by permission of the publisher.

in plants increased from below the detection limit to 133 ppm. Prince (1957) analyzed corn grown in four soil types: Sassafras sandy loam, Norton loam, Washington loam, and Cossayuna loam. Leaves accumulated up to 2.43 ppm cadmium; content increased with age of the plants. Cadmium concentrations in soil were not given.

4.2.2 Translocation, Distribution, and Accumulation

4.2.2.1 Crop Plants

4.2.2.1.1 Grains — Cadmium content of wheat from unpolluted areas of the eastern and midwestern United States was determined by Huffman and Hodgson (1973). Only 3 of 33 samples contained greater than 0.5 ppm, and the highest concentration was 0.71 ppm. In greenhouse studies, Haghiri (1973) found that wheat tops accumulated cadmium when the metal was applied to soil. Cadmium uptake by wheat from soil containing sewage sludge (cadmium content, 10 ppm) applied at rates of 0, 6.5, 19, 58, and 175 tons/ha was determined by Linnman et al. (1973). Applications at all but the highest rate increased cadmium uptake; maximum accumulation was 257 ± 9 ppb. The concentration of cadmium in whole wheat was 50 ppb, while that in bran was threefold higher at 148 ppb. In a similar study, Stenström and Lönsjö (1974) applied sludge (5.2 ppm cadmium) to several soils and determined the radiocadmium uptake by wheat; isotopic equilibration of added tracer with sludge cadmium is assumed. Uptake of tracer increased with increasing sludge addition, and 0.0033% of the cadmium originally in sludge was recovered in the mature crop. The authors estimated that when sludge containing 15 ppm cadmium was applied at a level of 1 metric ton/ha, it would increase the grain cadmium by 2.2%. They concluded that even a limited annual application of sludge may give rise to intolerable concentrations of cadmium in agricultural crops.

Other researchers have found that the cadmium content of grains increased by several hundred percent when the grains were grown on sewage sludge. Hinesly, Jones, and Ziegler (1972) observed a 240% increase in the cadmium content of corn grain at harvest, from 0.30 to 1.03 ppm, as from 0 to 1 in. (2.54 cm) of anaerobically digested sewage sludge was applied to the soil. Applications were made as frequently during the growing season as drying conditions of the sludge would permit. Dowdy and Larson (1975) found a 150% increase of cadmium in corn grain as sludge applications increased from 0 to 450 tons/ha. Sabey and Hart (1975) found a 190% increase in the cadmium content of wheat grain, from 0.066 to 0.190 ppm, as the sludge application rate increased from 0 to 100 metric tons/ha (dry weight). In this case, the municipal sewage sludge was added to a Truckton loamy sand in the field. For additional information on land application of sewage sludge, see Sections 4.2.2.1.2 and 7.5.

4.2.2.1.2 Vegetables and fruits — Haghiri (1973) compared root and foliar uptake of cadmium by soybean. Root uptake was 15 times more efficient, indicating that absorption of airborne cadmium is not as environmentally important as absorption from contaminated soils. Regardless of the method of application, the cadmium content of plant parts in this study decreased in the following order: stem > leaves > pods > beans. In the same study, the following uptake of cadmium by vegetables was observed on soil containing 10 ppm of the metal: lettuce (*Lactuca sativa*), 27.10 ppm; radish top

(*Raphanus sativus*), 16.13 ppm; celery stalk (*Apium graveolens*), 14.87 ppm; celery leaves, 10.77 ppm; green pepper (*Capsicum frutescens*), 6.28 ppm; and radish roots, 5.93 ppm. Williams and David (1973) also tested vegetable uptake of cadmium from soil (Table 4.7). The cadmium content of the edible portion was less than that of the nonedible portion in every case.

TABLE 4.7. CADMIUM CONTENT OF AND UPTAKE BY VEGETABLE SPECIES GROWN IN POT CULTURE

Species	Treatment	Cadmium content (ppm)		Total cadmium uptake (μg/pot)[a]	Added cadmium taken up[a] (%)	Moisture content of fresh edible material at harvest (%)
		Edible portion	Nonedible portion			
Peas	Nil	0.063	0.089	1.03		76
	Super[b]	0.233	0.265	3.77	3.3	
	CdCl$_2$[c]	0.238	0.555	6.58	2.8	
Beans	Nil	0.018	0.103	1.03		86
	Super	0.054	0.187	2.05	1.2	
	CdCl$_2$	0.071	0.252	2.77	0.9	
Tomato	Nil	0.074	0.214	3.10		93
	Super	0.157	0.596	9.46	7.6	
	CdCl$_2$	0.339	0.922	15.43	6.1	
Radish	Nil	0.136	0.237	1.47		91
	Super	0.656	1.020	6.78	6.3	
	CdCl$_2$	1.140	1.560	11.09	4.8	
Cabbage	Nil	0.076		1.08		80
	Super	0.220		3.48	2.9	
	CdCl$_2$	0.408		6.10	2.5	

[a] Plant tops only except for radish, which included roots.
[b] 2 g superphosphate, adding 85 μg cadmium per pot.
[c] CdCl$_2$, 200 μg per pot.

Source: Adapted from Williams and David, 1973, Table 10, p. 53. Reprinted by permission of the publisher.

Turner (1973) grew vegetables in nutrient solution containing 0 to 1.00 μg cadmium per milliliter. The cadmium content of all species tended to increase with increasing cadmium treatment. Tomato (*Lycopersicum esculentum*) was a high accumulator (0.3 to 158.0 ppm), while carrot (*Daucus carota*) was a low accumulator (0.2 to 2.2 ppm). John (1973) determined uptake of cadmium by vegetables from contaminated soil (Table 4.5). At the higher level of soil contamination, the mean cadmium concentration of plant parts in general was significantly higher than control values. The accumulation of cadmium in underground plant portions was dramatic (Figure 4.2).

Uptake of cadmium by radish was measured by John (1972). In unlimed soil, radish roots accumulated 174.3 ppm cadmium, which was 34.2 times the level in control plants. Radish tops accumulated 402.8 ppm. Lagerwerff

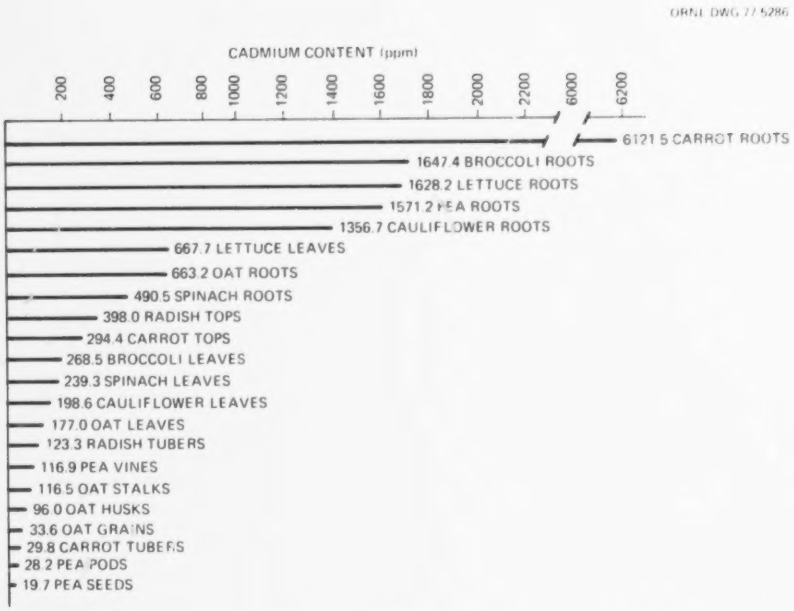


Figure 4.2. Cadmium content of plants grown in 200 mg cadmium per 1000 g soil. Source: Adapted from John, 1973, Figure 1, p. 14. Reprinted by permission of the publisher.

(1971) also found that cadmium accumulation was greater in radish tops than in roots. Turner (1973), measuring accumulation only in tops, found 144.2 ppm in plants grown in nutrient solutions containing 1.0 ppm cadmium. Kobayashi (1972) sampled vegetables at varying distances from a zinc refinery in Japan and concluded that root uptake of cadmium from contaminated soil appeared to be the principal source of plant cadmium, as opposed to foliar absorption from polluted air. Shacklette (1972) criticized this observation: "The vegetable analyses show relatively low amounts of cadmium in plant parts that have a low surface-to-volume ratio (asparagus stems, pumpkin, tomato, and eggplant fruit), in parts that are surrounded by protective outer structures (corn and bean seeds), and in plant parts that grow underground (potatoes, radish roots, taro potatoes, and turnip roots). Leaves, in contrast, have a high surface-to-volume ratio and were shown to usually contain much larger amounts of cadmium." Schroeder et al. (1967) tested a wide variety of plants and plant products for cadmium content. The cadmium content of vegetables ranged from below the detection limit to 0.45 ppm; fruits showed 0.01 to 0.14 ppm, and nuts 0.03 to 0.07 ppm (all values based on variable wet weights; cadmium content based on wet weights gives an indication of dietary cadmium intake).

Ross and Stewart (1969) sprayed apple trees with an aqueous solution of cadmium chloride (43 ppm cadmium) once a year for three years. Cadmium residues in foliage declined slowly from an initial concentration of about 25 μg/100 cm^2 to about 13 μg/100 cm^2 at the times of harvest. The initial cadmium concentration in apples was about 38 μg per ten apples, each of the first two years, whereas harvest residues were 95 μg in 1964 and 65 μg in

1965. In 1967, the initial cadmium content in fruit after spraying was 82 μg per ten apples; the content declined to 69 μg after a month but increased to 90 μg at harvest. The higher initial concentration in 1967 was due to increased apple size. Peels contained more cadmium than did pulp. Thus, cadmium residues persist in foliage during the growing season and may be translocated from foliage to fruit.

As in grains, vegetables and fruits grown in soil treated with sewage sludge also show increases in cadmium content. A 300% increase in the cadmium content of tomato fruit was observed by Dowdy and Larson (1975) as the applied sludge increased from 0 to 450 tons/ha. The increase in cadmium content of pea fruit was 33% and the cadmium content of carrots, radishes, and potatoes increased by 140%, 140%, and 90% respectively. Under the same conditions, the cadmium content of leaf lettuce increased 340%; Dowdy and Larson (1975) concluded that lettuce is an accumulator of metals.

4.2.2.1.3 Herbs — Gordee, Porter, and Langston (1960) exposed peppermint (*Mentha piperita*) plants to tracer cadmium. Plants were harvested after 4, 24, 48, and 72 hr. Remaining pots were than flushed with water to remove tracer and additional plants were collected at 7 and 14 days. Accumulation was not evident during the first 4 hr, but it increased throughout the remaining 68 hr. The 7- and 14-day harvests revealed that cadmium is translocated from older to newer plant growth and remains mostly in the vascular tissue.

Thus, crop plants normally accumulate only moderately high levels of cadmium. When background cadmium levels in soils are raised, however, plant uptake is significantly increased. Accumulated cadmium tends to be retained in the roots.

4.2.2.2 Noncrop Plants

4.2.2.2.1 Herbaceous plants — The consideration of mosses, lichens, and vascular plants in this section does not imply that methods of uptake in these plants are identical. Uptake in mosses is through sorption by rhizoids; in lichens, the fungal mycelium is responsible for uptake. Since mosses and lichens have no true roots, their methods of uptake should not be equated with that of vascular plants. Huckabee and Blaylock (1973) sampled mosses and grasses in a relatively uncontaminated area to determine near-background levels of cadmium. The mean cadmium content of the mosses *Dicranum* and *Plytrichum* was 0.421 and 0.383 ppm respectively. *Festuca* and *Andropogon* grasses averaged 0.279 and 0.210 ppm. Huffman and Hodgson (1973) determined the cadmium concentration in grasses from rural areas of the United States to be 0.17 ppm. Selected Alaskan grasses contained slightly less than 0.1 ppm (Kubota, Rieger, and Lazar, 1970).

Retention of ^{109}Cd by old field vegetation was studied by Matti, Witherspoon, and Blaylock (1975). Approximately 10% of $^{109}CdCl_2$ applied in simulated rainfall was retained after 24 to 48 hr. Seven days later, after three rainfalls, retention decreased to 7.3% of the total applied cadmium.

Cadmium content in grasses near four heavily traveled highways decreased with increased distance from the pavement (Lagerwerff and Specht, 1970).

TABLE 4.8. EXTRACTABLE CADMIUM AND ZINC CONTENTS OF ROADSIDE SOIL AND VEGETATION AS A FUNCTION OF DISTANCE FROM TRAFFIC AND DEPTH IN THE PROFILE[a]

Site	Metal	Distance from road (m)	Metal content (mg/kg dry wt)			
			Grass	Soil		
				0-5 cm	5-10 cm	10-15 cm
I. East of U.S. 1, near Plant Industry Station, Beltsville, Md.; 20,000 cars per day	Cadmium	8	1.25	0.65	0.44	0.23
		16	0.75	0.31	0.18	0.11
		32	0.58	0.17	0.12	0.08
	Zinc	8	64.5	192	98.2	30.3
		16	50.0	112	28.3	20.0
		32	41.2	43	22.1	16.2
II. East of Victory Road, Cincinnati, Ohio; 10,600 cars per day	Cadmium	8	0.63	1.04	0.80	0.54
		16	0.38	0.86	0.66	0.48
		32	0.25	0.66	0.52	0.30
	Zinc	8	92.4	55.5	27.3	3.5
		16	82.7	38.0	17.1	3.2
		32	72.5	17.4	7.9	1.3

[a] Average of duplicate samplings and analyses.

Source: Adapted from Lagerwerff and Specht, 1971, Table 1, p. 88. Reprinted by permission of the publisher.

Another study by Lagerwerff and Specht (1971) conducted near heavily traveled (20,000 cars per day) and moderately traveled (10,600 cars per day) highways had similar results (Table 4.8). Soil was also collected at 6 m (0.75 ppm cadmium) and 180 m (0.10 ppm cadmium) from the heavily used highway and seeded with bromegrass. Cultures were grown in either their natural environment or an artificial environment containing particulate-free air. The cadmium content of grass grown in natural air was 1.4 times higher than that in grass grown indoors in the low-cadmium soil; exposure to particulates of the natural environment increased the cadmium content 1.8 times.

Goodman and Roberts (1971) determined the cadmium content of moss (*Hypnum cupressiforme*) and grass (*Festuca rubra*) in polluted and unpolluted areas. The cadmium content of moss was 1.0 to 9.5 ppm in the polluted area and 1.0 to 1.8 ppm in the unpolluted area. Corresponding grass contents were 1.3 to 40.0 ppm and 0.7 to 0.8 ppm. Huckabee, Stella, and Olmstead (cited in Smith and Huckabee, 1973) studied ^{109}Cd uptake by mosses (*Eurhynchium hians*, *Brachythecium rivulare*, and *Sharpiella striatella*) and a grass (*Festuca elatior*). Both soluble and insoluble forms of cadmium were used; in both cases mosses retained at least ten times more cadmium than did grass (Figures 4.3 and 4.4).

Mosses were sampled at ten paired sites along highways by Shacklette (1972). One site for each pair was 45.7 to 61.0 m (15 to 20 ft) from the highway, the other 304.8 to 1828.8 m (100 to 600 ft) away. Samples near the highway contained a mean level of 5.6 ppm cadmium; those away from the highway averaged 5.2 ppm (difference not statistically significant). The author noted that dust present on the samples could not be completely removed by

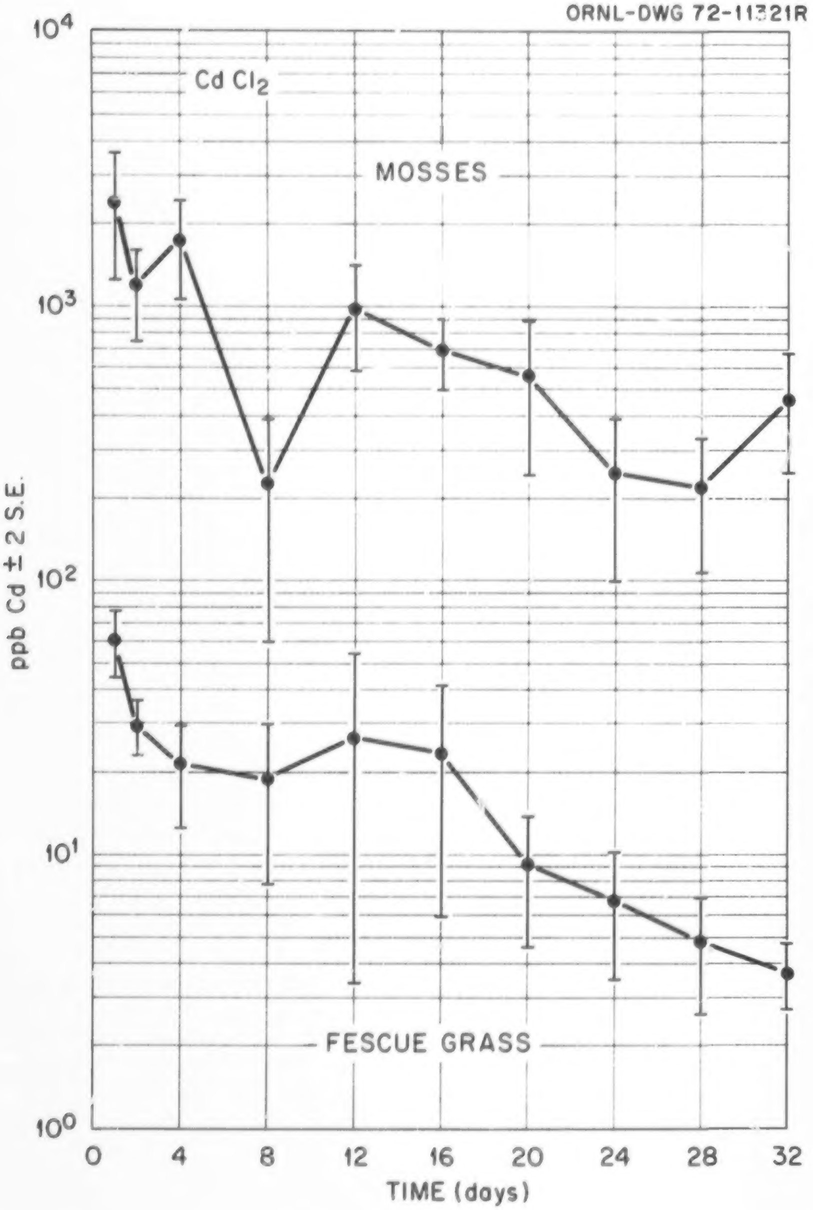


Figure 4.3. Uptake of ^{109}Cd by fescue grass and by three species of mosses following application as $CdCl_2$ in simulated rainfall. Source: Smith and Huckabee, 1973, Figure VI-11, p. 285.

washing and may have obscured larger differences. A U.S. Geological Survey study (Shacklette, 1972) which monitored Spanish moss (*Tillandsia usneoides*), a vascular plant and not a true moss, throughout the southeastern United States revealed that the cadmium content of the plant was related to the degree of air pollution in the sampling location.

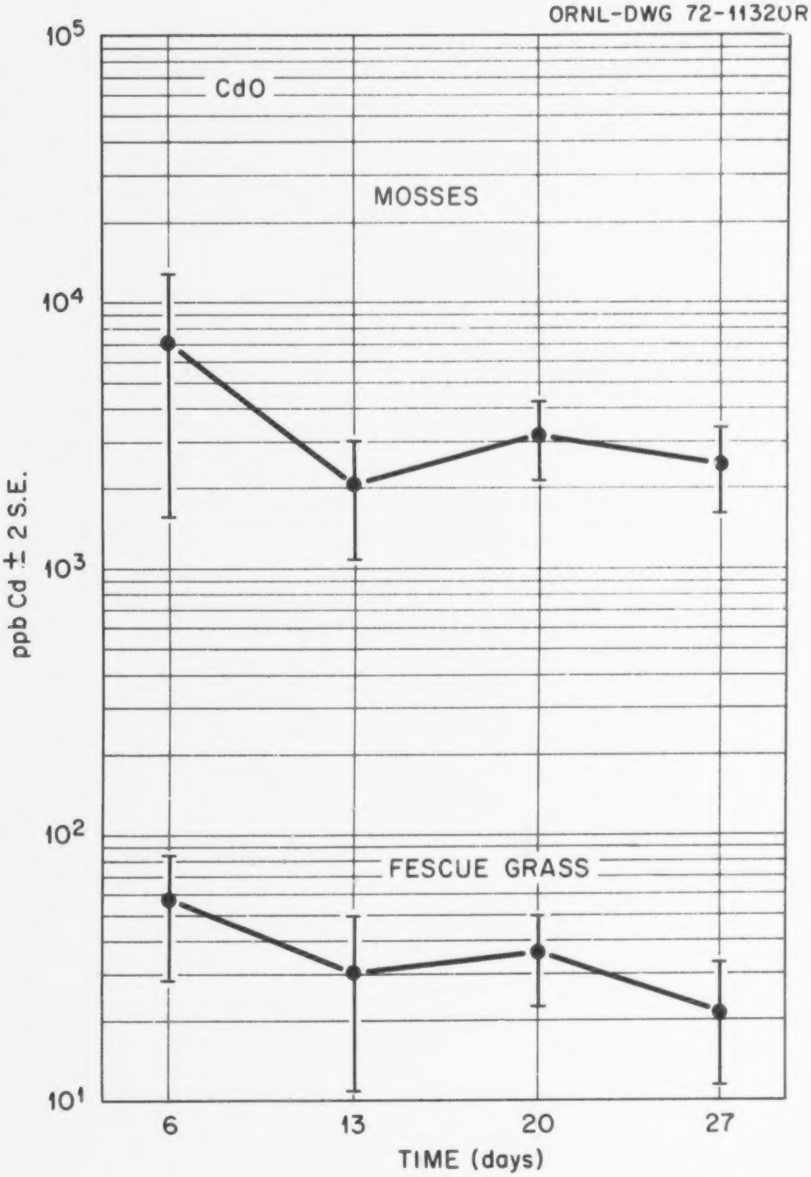


Figure 4.4. Uptake of ^{109}Cd by fescue grass and by three species of mosses following application as CdO in simulated rainfall. Source: Smith and Huckabee, 1973, Figure VI-12, p. 286.

Samples of moss (*Hypnum cupressiforme*) from a polluted area of Sweden contained 30 ppm cadmium; lichen (*Parmelia physodes*) contained 12 ppm (Tyler, 1972). Hairgrass (*Deschampsia flexuosa*) contained 7.6 ppm in leaves and 11 ppm in combined samples of roots and rhizomes. Tyler et al. (1973) found values of 0.30 g cadmium per hectare in mixed mosses and 0.36 g cadmium per hectare in lichens in a heath ecosystem.

Jaakkola, Takahashi, and Miettinen (1973) compared the cadmium content of lichens growing near a zinc smelter with those from an unpolluted area. Samples taken near the smelter approximately six months after smelting began contained about 1 ppm cadmium. Samples from the unpolluted area contained only about 0.1 to 0.2 ppm. Shacklette (1972) tested a species of fruticose lichen (*Cladonia rangiferina*) growing over a dolomite deposit and found approximately 0.19 ppm cadmium.

Lounamaa (1956) sampled vegetation in Finland to determine cadmium content. Fifteen of sixteen species of lichens growing on silicic rock contained 10 to 300 ppm cadmium in ash, while the remaining species contained 30 to 600 ppm in ash. Eight of nine species of mosses contained 1 to 10 ppm. The remaining mosses, collected in a contaminated area, contained 100 ppm cadmium in ash. For ferns, the highest cadmium concentration in fronds was 30 ppm, but an accumulation of as much as 100 ppm (mean = 22) was found in the ash of subterranean plant parts.

Cearley and Coleman (1973) exposed southern naiad (*Najas guadalupensis*) to $CdSO_4$. Exposure levels ranged from 0.0005 ± 0.0001 ppm (control) to 0.83 ± 0.12 ppm cadmium. As the exposure level increased, cadmium accumulation by the plants increased. After 21 days the plants subjected to the highest exposure contained 5429.3 ± 60.6 ppm cadmium compared with 7.1 ± 0.1 ppm in the control. Uptake of $CdNO_3$ by duckweed (*Lemna valdiviana*) and aquatic fern (*Salvinia natans*) from water culture was evaluated by Hutchinson and Czyrska (1972). As the cadmium content of the solution culture increased from 0.00 to 1.00 ppm, cadmium content of duckweed rose from 7.2 to 1475.5 ppm and that of aquatic fern rose from 2.1 to 6400 ppm.

4.2.2.2.2 Woody plants — Smith (1973) examined leaf and twig tissue from six tree species in New Haven, Connecticut (Table 4.9). As a check, 25

TABLE 4.9. CADMIUM CONTENT OF WOODY PLANTS GROWING IN NEW HAVEN, CONNECTICUT[a]

Species	Total trees sampled	Average distance from nearest street (m)	Tissue analyzed[b]		Cadmium content (ppm dry wt)	
			Organ	Number of trees	Mean and standard error	Range
Pin oak, *Quercus palustris* Muenchh.	24	1.4	Leaves	12	2.3 ± 0.2	1.5-3.0
			Twigs	12	2.3 ± 0.2	1.0-3.0
Sugar maple, *Acer saccharum* Marsh.	14	1.8	Leaves	7	1.0 ± 0.1	0.5-1.5
			Twigs	7	0.8 ± 0.1	0.5-1.5
Norway maple, *Acer platanoides* L.	64	2.0	Leaves	32	1.1 ± 0.1	0.5-2.0
			Twigs	32	0.7 ± 0.1	0.5-2.0
Eastern hemlock, *Tsuga canadensis* (L.) Carr.	16	3.7	Leaves	8	0.9 ± 0.1	0.5-1.0
			Twigs	8	1.2 ± 0.2	0.5-1.8
Yew, *Taxus* spp.	20	3.8	Leaves	10	1.2 ± 0.2	0.5-2.5
			Twigs	10	2.0 ± 0.4	0.5-4.1
Norway spruce, *Picea abies* (L.) Karst.	8	4.8	Leaves	4	0.7 ± 0.2	0.5-1.0
			Twigs	4	0.8 ± 0.2	0.5-1.0

[a] Branch samples were collected during the fall of 1970 approximately 2 m above the ground.
[b] Washed growth of the previous growing season only.

Source: Adapted from Smith, 1973, Table II, p. 633. Reprinted by permission of the publisher.

sugar maple samples were obtained from remote areas of Vermont and New Hampshire. Twigs and leaves of the remote samples contained 0 to 5 ppm cadmium; New Haven samples contained 0.5 to 4.1 ppm. Smith concluded that New Haven trees contained "normal" cadmium concentrations. Mixed tree species from Alaska were found to contain 0.4 to 0.5 ppm cadmium in leaves and twigs (Kubota, Rieger, and Lazar, 1970). Huckabee and Blaylock (1973) found that needles of eastern hemlock (*Tsuga canadensis*) contained a mean of 0.083 ppm cadmium; twigs contained 0.326 ppm. Tyler (1972) determined cadmium distribution in an area polluted by industry (Table 4.10) and concluded that an increase in heavy-metal deposition results in metal accumulation in the more exposed, permeable portions of the ecosystem such as mosses, lichens, humus, and rough bark of twigs. Anderson et al. (1974) reported the following values for samples from three deciduous species: leaves, 0.16 to 0.38 ppm; twigs, 0.05 to 0.93 ppm; branches, 0.03 to 0.39 ppm; bole, 0.2 to 0.33 ppm; and roots, 0.15 to 0.34 ppm. A coniferous species contained 0.48 ppm in leaves, 0.26 ppm in twigs, 0.30 ppm in branches, 0.21 ppm in bole, and 0.30 ppm in roots.

TABLE 4.10. CONCENTRATIONS OF CADMIUM IN COMPONENTS OF A SPRUCE FOREST IN CENTRAL SWEDEN POLLUTED BY A LOCAL INDUSTRIAL SOURCE

Species	Plant part	Cadmium content (ppm dry wt)	Enrichment ratio[a]
Spruce, *Picea abies*	Roots, <5 mm diam	2.7	6.8
	Roots, ≥5 mm diam	1.5	7.5
	Wood	<0.1	
	Bark	2.5	13
	Twigs		
	1st year	5.4	14
	2nd year	4.6	12
	3rd year	4.2	10
	4th year	3.3	7.5
	5th-7th year	2.7	6.0
	Needles		
	1st year	0.6	3.0
	2nd year	0.4	1.5
	3rd year	0.5	1.4
	4th year	0.5	1.4
	5th-7th year	1.0	2.4
	Needle liter	24	63
Cowberry, *Vaccinium vitis idaea*	Aboveground biomass	3.2	17
Bilberry, *Vaccinium myrtillus*	Aboveground biomass	4.4	13
Hair grass, *Deschampsia flexuosa*	Leaves	7.6	10
	Leaf litter	12	15
	Roots and rhizomes	11	16
Epiphytic lichens, *Parmelia physodes*		12	30
Mosses, *Hypnum cupressiforme*		30	33
Humus layer (raw humus)		44	40

[a] Ratio of cadmium concentration at this site to that at a similar site with no local deposition.

Source: Adapted from Tyler, 1972, Table 1, p. 59. Reprinted by permission of the publisher.

Elm (*Ulmus glabra*) leaves were analyzed from branches either facing or opposing a smelter (Little and Martin, 1972). Cadmium concentrations were higher in leaves from the sheltered side due to natural slowing of wind speed by the tree and, thus, greater metal precipitation. A comparison of washed and unwashed leaves revealed that only 28% of the total cadmium was removed by detergent washing, indicating partial cadmium penetration into the leaf. Smith (1973) found no significant difference between washed and unwashed leaves. Little (1973) found that about 67% of the cadmium content of oak (*Quercus robur*) leaves was removed after washing and boiling in deionized water, but only 15% to 20% was removed from willow (*Salix alba*) and hawthorn (*Crataegus monogyna*) leaves. The cadmium content of unwashed leaves collected about 400 m from a zinc and palladium smelter was 6.04 ± 0.98 ppm for hawthorn, 6.82 ± 1.33 ppm for oak, and 7.85 ± 1.24 ppm for willow.

Metal distribution in the aboveground portion of two heath ecosystems (*Calluna vulgaris* and *Erica tetralix*) was studied by Tyler et al. (1973). *Calluna* contained a total of 0.73 g cadmium per hectare; *Erica* contained 0.69 g/ha. The distribution of cadmium within each species is given in Table 4.11.

TABLE 4.11. DISTRIBUTION OF CADMIUM IN THE COMPONENTS OF ABOVEGROUND BIOMASS IN *CALLUNA* AND *ERICA* ECOSYSTEMS

Species	Plant part	Age	Cadmium content (g/ha)
	Calluna ecosystem		
Calluna vulgaris			0.73
	Short shoots and leaves	Current year	0.13
	Short shoots and leaves	Last year	0.19
	Short shoots and leaves	Older	0.02
	Long shoots and leaves	Current year	0.05
	Long shoots and leaves	Last year	0.08
	Long shoots and leaves	Older	0.26
Empetrum nigrum			0.10
Mosses and lichens			0.30
Other species			0.06
	Erica ecosystem		
Erica tetralix			0.69
	Leaves	Current year	0.11
	Leaves	Last year	0.09
	Leaves	Older	0.02
	Shoots	Current year	0.13
	Shoots	Last year	0.11
	Shoots	Older	0.23
Mosses and lichens			0.36
Other species			0.12

Source: Adapted from Tyler et al., 1973, Table 5, p. 261. Reprinted by permission of the publisher.

In a U.S. Geological Survey study (Shacklette, 1972), plants were sampled in six vegetation-type areas remote from major pollution sources in Missouri. The terminal 15 to 20 cm (6 to 8 in.) of stem was analyzed for cadmium content (Table 4.12). Soil samples from the area contained 1 ppm cadmium. According to Shacklette (1972), the data indicate that plant cadmium content is determined by the ability of the species involved to take up cadmium rather than by the amount of cadmium present in the soil. Lounamaa (1956) found that conifer needles usually contained less cadmium (slightly less than 10 ppm in ash) than twigs (about 30 ppm). Needles and twigs from trees growing on silicic rock contained more cadmium than those on ultrabasic or calcareous rock. For deciduous species, most leaf specimens contained less cadmium than did twigs. For "dwarf shrubs," 80% of the stem samples contained more cadmium than did the leaves.

Hanna and Grant (1962) determined the mineral composition of 12 species of woody plants growing on two different soil types regarded as having "normal" cadmium levels. The following cadmium contents were found in plants: *Acer rubrum*, 1.7 ppm (mean of four samples); *A. saccharinum*, 0.10 ppm; *A. saccharum*, 0.21 ppm; *Ilex opaca*, 5.2 ppm (mean of four samples); *Kalmia latifolia*, 0.43 ppm (mean of two samples); *Pieris japonica*, 1.2 ppm (mean of five samples); *Pinus strobus*, 0.9 ppm (mean of three samples); *Quercus palustris*, 2.4 ppm (mean of four samples); and *Tsuga canadensis*, 0.6 ppm (mean of two samples). After analysis of data for the two soil types, the authors concluded that greater variation occurred among different species growing on the same soil than in the same species growing on different soils. No data were presented on the soil cadmium levels. Fertilization was minimal and the only soil property reported was pH.

Data from a U.S. Geological Survey study of trace elements in plants growing near mineral deposits (Curtin and King, cited in Shacklette, 1972) are shown in Table 4.13. Stem ash generally contained more cadmium than leaf ash, indicating uptake from the soil rather than deposition from the air. If the source of cadmium had been airborne particles, stems, according to Shacklette (1972), would have contained less cadmium than leaves due to a lower surface-to-volume ratio. Cadmium in soil humus of the sampling area ranged from <1 to 10 ppm.

Blaylock et al. (1973) used cadmium in a tree inoculation study. Cadmium as $^{109}CdCl_2$ was promptly translocated from trunk (point of inoculation) to foliage of eastern red cedar (*Juniperus virginiana*). Approximately 65% of the cadmium reached the crown, of which 0.1% was leached by rain (20.8 cm total) over an eight-week period. Of the cadmium leached from the crown, 2.7% was absorbed by understory vegetation.

In summary, noncrop plants contain insignificant cadmium levels unless soil content exceeds background levels. Mosses tend to be cadmium accumulators and may be potential indicators of atmospheric concentrations. Woody plants accumulate cadmium in twigs. Generally, cadmium levels in noncrop plants do not appear to represent an environmental hazard.

TABLE 4.12. MEAN CADMIUM CONCENTRATIONS IN TREE, SHRUB, AND GRASS SAMPLES FROM SIX VEGETATION-TYPE AREAS IN MISSOURI

Species	Plant part	Number of samples	Cadmium in ash (ppm)			Percent ash			Arithmetic mean (ppm) cadmium converted to units of dry weight (ppm)
			AM[a]	GM[b]	GD[c]	AM[a]	GM[b]	GD[c]	
Big bluestem grass, *Andropogon gerardii* Vitman	Stem and leaf	3	0.5	0.5		6.0	5.6	1.45	0.03
Buckbush, *Symphoricarpos orbiculatus* Moench	Stem	149	15	12	2.05	2.7	2.6	1.21	0.41
Post oak, *Quercus stellata* Wang.	Stem	30	2.6	2.3	1.57	4.4	4.3	1.27	0.11
Red cedar, *Juniperus virginiana* L.	Stem and leaf	32	1.6	1.4	1.47	6.2	6.1	1.13	0.10
Shagbark hickory, *Carya ovata* (Mill.) K. Koch	Stem	16	24	19	1.96	5.3	5.1	1.29	1.27
Shortleaf pine, *Pinus echinata* Mill.	Stem and leaf	35	15	14	1.48	2.8	2.8	1.20	0.42
Smooth sumac, *Rhus glabra* L.	Stem	184	3.9	3.2	1.90	3.7	3.7	1.20	0.14
Sweet gum, *Liquidambar styraciflua* L.	Stem	26	8.4	6.2	2.17	4.9	4.6	1.37	0.41
White oak, *Quercus alba* L.	Stem	65	4.3	4.0	1.43	3.6	3.5	1.24	0.16
Willow oak, *Quercus phellos* L.	Stem	30	11	9.0	1.97	2.5	2.4	1.33	0.28
Winged sumac, *Rhus copallina* L.	Stem	29	3.3	2.8	1.84	3.3	3.2	1.22	0.11

[a] Arithmetic mean.
[b] Geometric mean.
[c] Geometric deviation.

Source: Adapted from Shacklette, 1972, Table 1, p. 7.

TABLE 4.13. MEAN CADMIUM CONCENTRATIONS AND ASH YIELDS IN TREE AND SHRUB SAMPLES FROM MINERALIZED AREAS IN COLORADO

Species	Leaves						Stems or branches					
	Cadmium in ash (ppm)			Percent ash		Geometric mean (ppm) cadmium converted to units of dry weight (ppm)	Cadmium in ash (ppm)			Percent ash		Geometric mean (ppm) cadmium converted to units of dry weight (ppm)
	Number of samples	GM[a]	GD[b]	GM[a]	GD[b]		Number of samples	GM[a]	GD[b]	GM[a]	GD[b]	
Colorado blue spruce, *Picea pungens* Engelm.	1	<1.0		3.3			1	2.0		1.5		0.03
Douglas fir, *Pseudotsuga menziesii* (Mirb.) Franco	15	2.0	1.77	3.6	1.28	0.07	15	7.3	2.47	2.7	1.36	0.20
Engelman's spruce, *Picea engelmannii* (Parry) Engelm.	5	1.1	1.36	4.9	1.30	0.05	5	6.4	1.56	3.5	1.25	0.22
Juniper, *Juniperus communis* ssp. *nana* Syme	14	2.5	2.17	4.4	1.16	0.11	14	5.5	1.66	4.7	1.13	0.26
Kinnikinnick, *Arctostaphylos uva-ursi* (L.) Spreng.	1	5.0		4.1		0.21	1	7.0		3.3		0.23
Limber pine, *Pinus flexilis* James	6	3.4	2.30	2.8	1.22	0.10	6	5.5	2.30	2.5	1.15	0.14
Lodgepole pine, *Pinus contorta* var. *latifolia* Engelm.	24	10.5	1.36	2.0	1.25	0.21	24	16.6	1.54	2.3	1.12	0.38
Moutain maple, *Acer glabrum* Torr.	5	1.6	3.06	5.3	1.12	0.09	5	4.9	2.00	3.5	1.26	0.17
Ponderosa pine, *Pinus ponderosa* var. *scopulorum* Engelm.	12	5.7	1.45	2.0	1.27	0.11	12	9.8	1.63	2.2	1.42	0.22
Snowberry, *Symphoricarpos occidentalis* Hook.	1	2.0		5.8		0.12	1	5.0		1.8		0.09
Sticky laurel, *Ceanothus velutinus* Dougl.	1	<2.0		4.4			1	1.0		3.0		0.03
Quaking aspen, *Populus tremuloides* Michx.	22	7.7	1.60	5.9	1.16	0.55	22	13.7	1.55	5.6	1.21	0.77
Wild gooseberry, *Ribes* sp.	2	1.2		5.9		0.07	2	1.7		4.7		0.08
Willow, *Salix* sp.	5	15.7	1.72	6.2	1.11	0.97	5	37.6	2.19	4.0	1.47	1.50

[a] Geometric mean.
[b] Geometric deviation.

Source: Adapted from Shacklette, 1972, Table [illegible], pp. [illegible].

4.2.3 Elimination — Gordee, Porter, and Langston (1960) determined that cadmium is not eliminated from peppermint. According to Shacklette (1972), there appears to be no natural means by which cadmium is eliminated from living plant tissue. No other references to cadmium elimination by plants were found.

4.3 EFFECTS

In general, the fact that a given cadmium concentration is toxic to plants depends on many largely unknown factors (Shacklette, 1972). Page, Bingham, and Nelson (1972), using nutrient solutions, found the yield of tops of beetroot, carrot, lettuce, radish, and Swiss chard to be essentially unaffected by cadmium levels of 0.01 to 0.10 ppm. Yield of tomato plants was severely reduced at 0.01 ppm. At a concentration of 1 ppm, cadmium reduced yields of tops of all species tested to 30% to 50% of controls. Chaney (1973) suggested that this work with nutrient solutions cannot easily be extrapolated to soils.

In greenhouse experiments using municipal sewage sludge moistened with cadmium sulfate, Bingham et al. (1975) observed that cadmium-sensitive plants such as spinach, soybean, curlycress, and lettuce were injured by soil cadmium levels of 3 to 4 ppm. In contrast, tomato and cabbage tolerated soil cadmium levels of approximately 170 ppm without apparent injury. Rice exhibited no ill effects at soil cadmium levels as high as 640 ppm. Thus, when comparing solution cultures (Page, Bingham, and Nelson, 1972) with soil cultures, critical cadmium concentrations may differ. In addition, results obtained in a particular soil are not necessarily applicable to other soils or soil mixes (Bingham et al., 1975).

Table 4.14 lists some phytotoxic effects exerted by cadmium on several economically important plants (Yopp, Schmid, and Holst, 1974). These data indicate that a general reduction in yield is the usual symptom of phytotoxic cadmium levels. Page, Bingham, and Nelson (1972) determined cadmium concentrations in nutrient solutions which resulted in a 50% growth reduction in some crops (Table 4.15). Significant differences are seen in the sensitivities of different species.

The effects of cadmium applied to soil on various crop yields were studied by John (1973). At a high rate of cadmium application, yields of most plant parts were depressed (Table 4.16). The yield of marketable portions (especially oat grain) was reduced more than that of roots. In other work, John (1972) found that as the amount of cadmium applied to soil increased from 0 to 100 ppm, yield of radish tops decreased from 1.59 to 1.00 g per pot and yield of roots decreased from 1.58 to 0.61 g per pot (number of plants per pot not given). Dry weight of corn plants grown in sandy soil decreased from 0.70 g per plant in the control to 0.25 g per plant when 562 ppm cadmium was added to the soil (Traynor and Knezek, 1973).

A cadmium concentration of 0.01 ppm in rooting solution inhibited frond production of duckweed (Hutchinson and Czyrska, 1972). A concentration of 0.05 ppm reduced growth 35% over a one-week period; at 0.5 and 1.0 ppm cadmium, plants ceased growth and eventually died. Similar results occurred

TABLE 4.14. PHYTOTOXIC EFFECTS EXERTED BY CADMIUM ON SEEDLING PLANTS OF ECONOMIC IMPORTANCE IN ILLINOIS[a]

Plant	Growing medium	Minimum phytotoxic concentration of cadmium (ppm)	Phytotoxic symptoms
Soybean	Defined soil type	2.5	Reddish brown veins in youngest trifoliate leaves; growth retardation
Winter wheat	Defined soil type	2.5	General growth retardation
Lettuce	Defined soil type	2.5	General growth retardation
Radish	Defined soil type	2.5	General growth retardation
Celery	Defined soil type	2.5	General growth retardation
Green pepper	Defined soil type	2.5	General growth retardation
Beet (root)	Defined nutrient medium	1.0	General growth retardation
Swiss chard	Defined nutrient medium	0.1	General growth retardation
Tomato	Defined nutrient medium	0.1	General growth retardation
Carrot	Defined nutrient medium	1.0	General growth retardation

[a] Entire plant affected in all cases.

Source: Adapted from Yopp, Schmid, and Holst, Table I, p. 74. Reprinted by permission of the publisher.

TABLE 4.15. CADMIUM CONCENTRATIONS IN NUTRIENT SOLUTIONS PRODUCING 50% GROWTH REDUCTION IN SOME CROPS

Plant	Cadmium concentration (μg/ml)		Concentration of cadmium in leaf at 50% growth depression[a] (μg/g)	Visible symptoms associated with 50% growth depression
	Range tested	Concentration producing 50% growth depression		
Cabbage	0-10	9.0	800	No specific symptoms were associated with reduced growth.
Tomato	0-10	4.8	570	Chlorosis
Barley	0-10	5.6	120	No specific symptoms were associated with reduced growth.
Pepper	0-10	2.0	160	Chlorosis
Sweet corn	0-10	1.2	230	Reddish orange coloration of leaf margin, necrosis, and chlorosis
Lettuce	0-10	0.9	320	Chlorosis
Red beet	0-10	0.2	290	Wilting
Field bean	0-1.0	0.2	22	Wilting
Turnip	0-1.0	0.2	290	Chlorosis

[a] Oven dry weight, 70°C.

Source: Adapted from Page, Bingham, and Nelson, Table 2, p. 290.

TABLE 4.16. EFFECT OF CADMIUM CONTAMINATION OF SOIL ON YIELD OF VARIOUS PARTS OF EIGHT CROPS

Crop	Plants per pot	Growth period (days)	Plant part	Dry weight (g/pot)[a]		
				Control	40 mg Cd per kilogram of soil	200 mg Cd per kilogram of soil
Lettuce	6	35	Leaves	17.50 *a*	18.82 *a*	1.55 *b*
			Roots	0.91 *a*	0.99 *a*	0.36 *a*
Spinach	8	55	Leaves	10.96 *a*	0.39 *b*	0.16 *b*
			Roots	0.51 *a*	0.02 *b*	0.02 *b*
Broccoli	3	60	Leaves	95.93 *a*	122.50 *a*	35.20 *b*
			Roots	2.46 *a*	4.08 *a*	2.09 *a*
Cauliflower	3	70	Leaves	103.67 *a*	100.83 *a*	3.17 *b*
			Roots	1.78 *a*	1.47 *a*	0.17 *b*
Pea	3	95	Seeds	27.45 *a*	16.98 *a b*	0.21 *b*
			Pods	10.59 *a*	7.45 *a*	0.84 *b*
			Vines	19.64 *a*	14.43 *a*	2.47 *b*
			Roots	1.98 *a*	1.08 *a*	0.56 *b*
Oat	12	100	Grains	42.57 *a*	27.28 *b*	18.37 *b*
			Husks	5.56 *b*	8.31 *a*	6.85 *a b*
			Leaves	20.84 *a*	21.50 *a*	18.72 *a*
			Stalks	29.90 *a*	39.17 *a*	23.28 *a*
			Roots	3.75 *a*	3.18 *a*	2.88 *a*
Radish	8	45	Tops	8.24 *a*	6.25 *b*	1.47 *c*
			Tubers	7.10 *a*	5.12 *a*	0.48 *b*
Carrot	10	130	Tops	18.95 *a*	16.92 *a*	1.50 *b*
			Tubers	41.72 *a*	38.27 *a*	1.52 *b*
			Roots	0.96 *a*	0.81 *a*	0.13 *b*

[a] Values followed by the same letter do not differ significantly at the 5% level.

Source: Adapted from John, 1973, Table 2, p. 11. Reprinted by permission of the publisher.

with aquatic fern. Cearley and Coleman (1973) reported that southern naiad exhibited toxicity symptoms (reduced chlorophyll content, turgor, and stolon development) as cadmium concentrations were raised from 0.007 to 0.83 ppm.

Haghiri (1973) noted that cadmium was toxic to wheat at the lowest concentration tested (2.5 ppm). Imai and Siegel (1973) reported that growth of red kidney bean embryos was inhibited by concentrations greater than 3 ppm in nutrient media. Broad beans (*Vicia faba*) lost turgor 2 to 3 hr after exposure to 0.5×10^{-3} M or 1×10^{-3} M concentrations of $CdCl_2$ (Nosseir, 1970). The cadmium altered normal ion absorption and release. Similar but less pronounced effects were recorded for kidney beans.

Puerner and Siegel (1972) found that 10 ppm cadmium added to rooting media of cucumbers (*Cucumis sativus*) produced severe root damage and induced shoots to spiral 180° around their own axes. Cadmium chloride (5×10^{-4} M)

applied to endocarp surfaces of excised immature tissue or intact seedlings of peas (*Pisum sativum*) resulted in an increased production of pisatin (Hadwiger, von Broembsen, and Eddy, 1973), a phytoalexin produced in response to certain plant pathogens. A concentration of 6 x 10^{-4} *M* cadmium increased RNA synthesis. Cadmium acetate applied to tobacco leaves results in a decrease in leaf dry weight and leaf sap phosphorus (Verma, 1971).

Huang, Bazzaz, and Vanderhoef (1974) found that soybeans grown in nutrient solution were sensitive to cadmium. Concentrations as low as 18 micromoles reduced fresh weight by approximately 35% and caused a 71% decline in nitrogenase activity. A lower nodule carbohydrate content caused by inhibition of photosynthesis was also noted. Cadmium nitrate is a potent inhibitor of photosynthesis in isolated chloroplasts of maize (Bazzaz, 1974). Absorption and fluoresence studies revealed that cadmium results in a 20% to 30% decrease in the chlorophyll A to chlorophyll B ratio. Bazzaz, Carlson, and Rolfe (1974) determined that a leaf tissue concentration of 0.93 ppm cadmium reduced photosynthesis by 50% in detached sunflower (*Helianthus annuus*) leaves. The primary mode of action was interference with stomatal function.

In conclusion, cadmium levels which affect plant metabolism are highly variable and virtually unknown for field crops. In controlled experiments cadmium has produced decreases in plant yield. Major effects reported in the literature include (1) altered ion absorption and release, (2) altered stomatal function, (3) reduced chlorophyll content, (4) reduced cell turgor, and (5) reduced photosynthesis. Generally, plants exposed to normal background levels are not known to experience any major inhibitory effects. Plants near cadmium sources, however, may contain abnormal cadmium levels detrimental to growth.

SECTION 4

REFERENCES

1. Anderson, S. H., A. W. Andren, C. F. Baes, III, G. J. Dodson, W. F. Harris, G. S. Henderson, D. E. Reichle, J. D. Story, R. I. Van Hook, W. Van Winkle, and A. J. Yates. 1974. Environmental Monitoring of Toxic Materials in Ecosystems. In: Ecology and Analysis of Trace Contaminants. ORNL/NSF/EATC-6, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 95-139.

2. Bazzaz, M. B. 1974. Effects of Cadmium Nitrate on Spectral Characteristics and Light Reactions of Chloroplasts. Environ. Lett. 6(1):1-12.

3. Bazzaz, F. A., R. W. Carlson, and G. L. Rolfe. 1974. The Effect of Heavy Metals on Plants: Part I. Inhibition of Gas Exchange in Sunflower by Pb, Cd, Ni and Tl. Environ. Pollut. (Great Britain) 7:241-246.

4. Bingham, F. T., A. L. Page, R. J. Mahler, and T. J. Ganje. 1975. Growth and Cadmium Accumulation of Plants Grown on a Soil Treated with a Cadmium-enriched Sewage Sludge. J. Environ. Qual. 4(2):207-211.

5. Blaylock, B. G., R. A. Goldstein, J. W. Huckabee, S. Janzen, C. Matti, R. G. Olmstead, M. Slawsky, R. A. Stella, and J. P. Witherspoon. 1973. Ecology of Toxic Metals. In: Ecology and Analysis of Trace Contaminants. ORNL/NSF/EATC-1, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 121-160.

6. Cearley, J. E., and R. L. Coleman. 1973. Cadmium Toxicity and Accumulation in Southern Naiad. Bull. Environ. Contam. Toxicol. 9(2):100-102.

7. Chaney, R. L. 1973. Crop and Food Chain Effects of Toxic Elements in Sludges and Effluents. In: Proceedings of the Joint Conference on Recycling Municipal Sludges and Effluents on Land. U.S. Environmental Protection Agency, U.S. Department of Agriculture, and National Association of State Universities and Land-Grant Colleges. pp. 129-141.

8. Cutler, J. M., and D. W. Rains. 1974. Characterization of Cadmium Uptake by Plant Tissue. Plant Physiol. 54:67-71.

9. Dowdy, R. H., and W. E. Larson. 1975. The Availability of Sludge-borne Metals to Various Vegetable Crops. J. Environ. Qual. 4(2): 278-282.

10. Francis, C. W., and S. G. Rush. 1973. Factors Affecting Uptake and Distribution of Cadmium in Plants. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 7:75-81.

11. Goodman, G. T., and T. M. Roberts. 1971. Plants and Soils as Indicators of Metals in the Air. Nature (Great Britain) 231:287-292.

12. Gordee, R. S., C. L. Porter, and R. G. Langston. 1960. Uptake and Distribution Studies of Cadmium 115-m in Peppermint. Proc. Am. Soc. Hortic. Sci. 75:525-528.

13. Hadwiger, L. A., S. von Broembsen, and R. Eddy, Jr. 1973. Increased Template Activity in Chromatin from Cadmium Chloride Treated Pea Tissues. Biochem. Biophys. Res. Commun. 50(4):1120-1128.

14. Haghiri, F. 1973. Cadmium Uptake by Plants. J. Environ. Qual. 2(1):93-96.

15. Haghiri, F. 1974. Plant Uptake by Cadmium as Influenced by Cation Exchange Capacity, Organic Matter, Zinc, and Soil Temperature. J. Environ. Qual. 3(2):180-183.

16. Hanna, W. J., and C. L. Grant. 1962. Spectrochemical Analysis of the Foliage of Certain Trees and Ornamentals for 23 Elements. Bull. Torrey Bot. Club 89(5):293-302.

17. Hinesly, T. D., R. L. Jones, and E. L. Ziegler. 1972. Effects on Corn by Applications of Heated Anaerobically Digested Sludge. Compost Sci. 13(4):26-30.

18. Huang, C., F. A. Bazzaz, and L. N. Vanderhoef. 1974. The Inhibition of Soybean Metabolism by Cadmium and Lead. Plant Physiol. 54:122-124.

19. Huckabee, J. W., and B. G. Blaylock. 1973. Transfer of Mercury and Cadmium from Terrestrial to Aquatic Ecosystems. Adv. Exp. Med. Biol. 40:125-160.

20. Huffman, E.W.D., Jr., and J. F. Hodgson. 1973. Distribution of Cadmium and Zinc/Cadmium Ratios in Crops from 19 States East of the Rocky Mountains. J. Environ. Qual. 2(2):289-291.

21. Hutchinson, T. C., and H. Czyrska. 1972. Cadmium and Zinc Toxicity and Synergism to Floating Aquatic Plants. In: Water Pollution Research in Canada 1972. University of Toronto, Toronto, Ontario, Canada. pp. 59-65.

22. Imai, I., and S. M. Siegel. 1973. A Specific Response to Toxic Cadmium Levels in Red Kidney Bean Embryos. Physiol. Plant. (Sweden) 29:118-120.

23. Jaakkola, T., H. Takahashi, and J. K. Miettinen. 1973. Cadmium Content in Sea Water, Bottom Sediment, Fish, Lichen, and Elk in Finland. Environ. Qual. Saf. 2:230-237.

24. John, M. K. 1972. Uptake of Soil-Applied Cadmium and Its Distribution in Radishes. Can. J. Plant Sci. (Canada) 52:715-719.

25. John, M. K. 1973. Cadmium Uptake by Eight Food Crops as Influenced by Various Soil Levels of Cadmium. Environ. Pollut. (Great Britain) 4:7-15.

26. John, M. K., H. H. Chuah, and C. J. VanLaerhoven. 1972. Cadmium Contamination of Soil and Its Uptake by Oats. Environ. Sci. Technol. 6(6):555-557.

27. John, M. K., C. J. VanLaerhoven, and H. H. Chuah. 1972. Factors Affecting Plant Uptake and Phytoxicity of Cadmium Added to Soils. Environ. Sci. Technol. 6(12):1005-1009.

28. Kobayashi, J. 1972. Air and Water Pollution by Cadmium Lead and Zinc Attributed to the Largest Zinc Refinery in Japan. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 5:117-128.

29. Kubota, J., S. Rieger, and V. A. Lazar. 1970. Mineral Composition of Herbage Browsed by Moose in Alaska. J. Wildl. Manage. 34(3):565-569.

30. Lagerwerff, J. V. 1971. Uptake of Cadmium, Lead and Zinc by Radish from Soil and Air. Soil Sci. 111(2):129-133.

31. Lagerwerff, J. V., and G. T. Biersdorf. 1972. Interaction of Zinc with Uptake and Translocation of Cadmium in Radish. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 5:515-522.

32. Lagerwerff, J. V., and A. W. Specht. 1970. Contamination of Roadside Soil and Vegetation with Cadmium, Nickel, Lead, and Zinc. Environ. Sci. Technol. 4:583-586.

33. Lagerwerff, J. V., and A. W. Specht. 1971. Occurrence of Environmental Cadmium and Zinc, and Their Uptake by Plants. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 4:85-93.

34. Linnman, L., A. Andersson, K. O. Nilsson, B. Lind, T. Kjellström, and L. Friberg. 1973. Cadmium Uptake by Wheat from Sewage Sludge Used as a Plant Nutrient Source. Arch. Environ. Health 27:45-47.

35. Little, P. 1973. A Study of Heavy Metal Contamination of Leaf Surfaces. Environ. Pollut. (Great Britain) 5:159-172.

36. Little, P., and M. H. Martin. 1972. A Survey of Zinc, Lead and Cadmium in Soil and Natural Vegetation around a Smelting Complex. Environ. Pollut. (Great Britain) 3:241-254.

37. Lounamaa, J. 1956. Trace Elements in Plants Growing Wild on Different Rocks in Finland. Ann. Bot. Soc. Zool. Bot. Fenn. "Vanamo" (Finland) 29:1-195.

38. Matti, C. S., J. P. Witherspoon, and B. G. Blaylock. 1975. Cycling of Mercury and Cadmium in an Old Field Ecosystem during One Growing Season. ORNL/NSF/ETAC-10, Oak Ridge National Laboratory, Oak Ridge, Tenn. 75 pp.

39. Nosseir, M. A. 1970. Rhythmicity in the Absorption and Release of Ions by Broad and Kidney Beans. Adv. Front. Plant Sci. (India) 25:121-128.

40. Page, A. L., F. T. Bingham, and C. Nelson. 1972. Cadmium Absorption and Growth of Various Plant Species as Influenced by Solution Cadmium Concentration. J. Environ. Qual. 1(3):288-291.

41. Prince, A. L. 1957. Influence of Soil Types on the Mineral Composition of Corn Tissues as Determined Spectrographically. Soil Sci. 83:399-405.

42. Puerner, N. J., and S. M. Siegel. 1972. The Effects of Mercury Compounds on the Growth and Orientation of Cucumber Seedlings. Physiol. Plant. (Sweden) 26:310-312.

43. Reuss, J., H. L. Dooley, and W. Griffis. 1976. Plant Uptake of Cadmium from Phosphate Fertilizer. EPA-600/3-76-053, U.S. Environmental Protection Agency, Corvallis, Ore. 37 pp.

44. Ross, R. G., and D.K.R. Stewart. 1969. Cadmium Residues in Apple Fruit and Foliage following a Cover Spray of Cadmium Chloride. Can. J. Plant Sci. (Canada) 49:49-52.

45. Sabey, B. R., and W. E. Hart. 1975. Land Application of Sewage Sludge: I. Effect on Growth and Chemical Composition of Plants. J. Environ. Qual. 4(2):252-256.

46. Schroeder, H. A., A. P. Nason, I. H. Tipton, and J. J. Balassa. 1967. Essential Trace Metals in Man: Zinc; Relation to Environmental Cadmium. J. Chronic Dis. (Great Britain) 20:179-210.

47. Shacklette, H. T. 1972. Cadmium in Plants. U.S. Geological Survey Bulletin 1314-G, U.S. Government Printing Office, Washington, D.C. 28 pp.

48. Smith, R. H., and J. W. Huckabee. 1973. Ecological Studies of the Movement, Fate, and Consequences of Cadmium. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 278-322.

49. Smith, W. H. 1973. Metal Contamination of Urban Woody Plants. Environ. Sci. Technol. 7(7):631-636.

50. Stenström, T., and H. Lönsjö. 1974. Cadmium Availability to Wheat: A Study with Radioactive Tracers under Field Conditions. Ambio (Norway) 3(2):87-90.

51. Stenström, T., and M. Vahter. 1974. Cadmium and Lead in Swedish Commercial Fertilizers. Ambio (Norway) 3(2):91-92.

52. Traynor, M. F., and B. D. Knezek. 1973. Effects of Nickel and Cadmium Contaminated Soils on Nutrient Composition of Corn Plants. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 7:83-87.

53. Turner, M. A. 1973. Effect of Cadmium Treatment on Cadmium and Zinc Uptake by Selected Vegetable Species. J. Environ. Qual. 2(1):118-119.

54. Tyler, G. 1972. Heavy Metals Pollute Nature, May Reduce Productivity. Ambio (Norway) 1(2):52-59.

55. Tyler, G., C. Gullstrand, K. Holmquist, and A. Kjellstrand. 1973. Primary Production and Distribution of Organic Matter and Metal Elements in Two Heath Ecosystems. J. Ecol. (Great Britain) 61(1):251-268.

56. Van Hook, R. I., Jr., B. G. Blaylock, E. A. Bondietti, C. W. Francis, J. W. Huckabee, D. E. Reichle, F. H. Sweeton, and J. P. Witherspoon. 1974. Radioisotope Techniques to Evaluate the Environmental Behavior of Cadmium. In: Comparative Studies of Food and Environmental Contamination. IAEA-SM-175/22, International Atomic Energy Agency, Vienna. pp. 23-42.

57. Verma, H. N. 1971. Some Metabolic Changes Induced by Cadmium Acetate in Healthy and TMV Infected Tobacco. Indian Phytopathol. (India) 24:354-358.

58. Williams, C. H., and D. J. David. 1973. The Effect of Superphosphate on the Cadmium Content of Soils and Plants. Aust. J. Soil Res. (Australia) 11:43-56.

59. Yopp, J. H., W. E. Schmid, and R. W. Holst. 1974. Cadmium. In: Determination of Maximum Permissible Levels of Selected Chemicals That Exert Toxic Effects on Plants of Economic Importance in Illinois. Illinois Institute for Environmental Quality, Chicago. pp. 69-78.

60. Yost, K. J., W. Bruns, J. E. Christian, F. M. Clikeman, R. B. Jacko, D. R. Masarik, W. W. McFee, A. W. McIntosh, J. E. Newman, R. I. Pietz, and A. M. Zimmer. 1973. The Environmental Flow of Cadmium and Other Trace Metals, Vol. I. Purdue University, West Lafayette, Ind. 42 pp.

SECTION 5

EFFECTS OF CADMIUM ON AQUATIC ANIMALS AND BIRDS

5.1 SUMMARY

Fish kills and excessive levels of pollutants in seafood illustrate the disposal problem for industrial and other wastes. Animals are valuable indicators of environmental pollution, and periodic sampling of trace metals in fish from waters into which industrial wastes are dumped helps to evaluate pollution control measures.

In fish, cadmium is absorbed through gills and/or the digestive tract. Concentration of the metal in tissues resembles that in other species (Section 6): levels are highest in kidneys and liver and low in muscle. Scallops concentrate cadmium from seawater to a great extent — by a factor of about 100 in one study. Shellfish, in general, accumulate cadmium to a greater extent than fish. Accumulation of cadmium may continue over the organism's lifetime. In bass and bluegill, equilibrium is reached between water and tissue cadmium concentrations.

Toxicity of cadmium can vary with water hardness and temperature and presence of other metals. A cadmium concentration of 0.008 to 0.010 ppm killed 50% of rainbow trout after seven days in hard water at a temperature of 11.0°C to 12.5°C. Cadmium is generally more toxic to fiddler crabs at higher temperatures and lower salinities.

Birds also take up cadmium mainly by ingestion. About 8% of ingested cadmium was absorbed by chipping sparrows. The tissue distribution is similar to that in other organisms mentioned, with kidneys and liver containing the highest concentrations. Cadmium levels in birds are generally low; 72% of starling samples collected across the United States contained less than 0.05 ppm whole-body weight. Levels in Cooper's hawk eggs were generally low but were significantly higher in eggs from unsuccessful nests than from successful ones. Toxic effects in birds are similar to those in other species and include growth retardation, anemia, and testicular hypoplasia.

5.2 FISH AND OTHER AQUATIC ORGANISMS

5.2.1 Metabolism

5.2.1.1 Uptake and Absorption — Fish and other aquatic animals absorb cadmium through gills and/or through the gastrointestinal tract. Because in the natural situation it is impossible to differentiate between the two routes of uptake, they are considered together in this section.

Mount and Stephan (1967) exposed bluegills to cadmium in a flow-through system for 90 days. They reported that there was substantial accumulation of cadmium in kidney, liver, gill, and gut, with lesser accumulation in spleen and none in muscle or bone.

Eisler, Zaroogian, and Hennekey (1972) examined cadmium uptake by adult mummichogs, scallops, and oysters and by subadult lobsters. All species were collected from Rhode Island waters and acclimated for 28 days in flowing, unfiltered seawater at 16°C to 20°C before separation into control and experimental groups. Cadmium (0.01 ppm as $CdCl_2$) was then added to the seawater of the experimental group for 21 days. Control seawater contained 0.001 ppm cadmium. As shown in Table 5.1, some marine organisms accumulate cadmium even when it is present at a very low concentration in water.

TABLE 5.1. CADMIUM UPTAKE BY MARINE ORGANISMS

Species	Cadmium content (ppm wet wt)		Increase over control (%)
	Experimental	Control	
Crassostrea virginica (whole system meats)	1.49	0.33	352
Aquipecten irradians (scallop, sum of soft parts	2.46	1.15	114
Homarus americanus (lobster, sum of parts)	0.72	0.51	41
Fundulus heteroclitus (whole mummichog)	0.48	0.33	45

Source: Adapted from Eisler, Zaroogian, and Hennekey, 1972, Table 1, p. 1368. Reprinted by permission of the Journal of the Fisheries Research Board of Canada.

Cearley and Coleman (1974) reported that cadmium uptake by largemouth bass and bluegills established a steady state between water and tissue concentrations after two months. Maximum whole-body burden of cadmium in bass was eightfold and fifteenfold greater than controls at exposures of 0.008 and 0.08 ppm cadmium respectively. Because the fish continued to grow, the absolute amount of cadmium per whole fish continued to increase although the amount per unit weight remained constant.

Kumada et al. (1973) found that maximum cadmium concentrations in rainbow trout exposed to cadmium levels in water of up to 0.0048 ppm occurred after 10 to 20 weeks. Cadmium concentrations as high as 300 times that in water were achieved. Total body content of cadmium decreased only moderately in ten weeks following transfer to cadmium-free water. These results are shown in Table 5.2.

TABLE 5.2. DISTRIBUTION OF CADMIUM IN ORGANS OF RAINBOW TROUT REARED IN CADMIUM SOLUTION (0.0043 ppm)

Time elapsed (weeks)[a]	Tissue	Cadmium content (ppm)		
		Wet sample	Dry sample	Ash
30	Muscle	0.12	0.40	5.2
	Skin	0.59	1.8	18
	Liver	7.3	37	350
	Kidney	20	100	1100
	Digestive tract			
	Gill	9.5	38	240
	Bone	0.85	1.1	1.2
	Residue	1.6	3.1	31
40	Muscle	0.04	0.14	2.2
	Skin	0.32	0.84	11
	Liver	4.4	19	230
	Kidney	16	75	860
	Gill	0.63	2.1	11
	Bone	0.38	0.66	0.80
	Residue	1.3	1.6	27

[a] The fish were exposed to cadmium for 30 weeks and then put in flowing water free of cadmium for 10 weeks.

Source: Adapted from Kumada et al., 1973, Table 3, p. 162. Reprinted by permission of the publisher.

Rainbow trout fed cadmium in food (up to 100 ppm of diet) also had increased levels of cadmium in their tissues (Kumada et al., 1973). On a dry weight basis, the maximum whole-body cadmium concentration was less than one-tenth that in the diet. Liver and kidney contained the most cadmium. Unlike cadmium absorbed from water, cadmium absorbed from the diet was rapidly eliminated when the fish were switched to cadmium-free diets. In whole fish, cadmium levels dropped by 90% on a relative basis (parts per million) and by 80% on an absolute basis in eight weeks after cadmium-free diets were started. Kidney levels remained high, suggesting a possible role of these organs in cadmium excretion.

Eaton (1974) reported information regarding the sites of deposition of cadmium in bluegills exposed for 11 months. He found (Table 5.3) that at each of the cadmium concentrations examined, the highest cadmium residues were found in the liver. Cadmium concentrations in intestine and cecum were somewhat lower and also increased with water concentration. In contrast, kidney cadmium did not increase with exposure levels.

TABLE 5.3. CADMIUM CONCENTRATIONS IN BLUEGILL TISSUES AFTER AN 11-MONTH CHRONIC EXPOSURE

Measured cadmium concentration in water (ppm)	Number of fish examined[a]	Cadmium content (ppm)[b]			
		Gill	Intestine and cecum	Liver	Kidney
0.0023 (control)	18 L	Trace[c]	Trace[c]	Trace[d]	Trace[d]
	0 D				
0.031	18 L	34 (22-51)	73 (49-99)	201 (138-298)	188 (96-305)
	0 D				
0.080	9 L	37 (18-60)	140 (73-327)	325 (218-472)	213 (170-293)
	4 D	21 (3-47)	210 (104-378)	343 (126-614)	157 (44-275)
0.239	2 L	45 (38 and 53)	349 (274 and 424)	538 (496 and 581)	218 (169 and 268)
	6 D	23 (15-26)	302 (113-461)	335 (146-422)	85 (7-208)
0.757	0 L				
	16 D	40 (22-73)	173 (67-336)	275 (114-458)	125 (42-344)
2.14	0 L				
	18 D	80 (38-211)	377 (131-756)	440 (172-755)	145 (67-295)

[a] Fish that lived to the end of the test (L) or died during it (D).

[b] Mean concentration with range in parentheses.

[c] Less than 5 ppm.

[d] Less than 10 ppm.

Source: Adapted from Eaton, 1974, Table 4, p. 733. Reprinted by permission of the publisher.

5.2.1.2 Normal Levels — Because aquatic organisms take up cadmium and fish and shellfish are an important food source for many people, they potentially could contribute to the human body burden of cadmium. Fish from cadmium-contaminated waters may have higher cadmium levels in tissues and organs than fish from uncontaminated water; however, the results are not always clear-cut.

For example, fish from the Holston River in Knox County, Tennessee, taken near the outfall of a zinc beneficiation mill contained 0.023 to 0.23 ppm cadmium in muscle and 0.47 to 2.9 ppm cadmium in kidneys (wet weight), while fish from a nearby lake with no known source of cadmium except normal background sources had 0.02 to 0.056 ppm cadmium in muscle and 0.2 to 2.0 ppm cadmium in kidneys (Huckabee et al., 1971, personal communication cited in Smith and Huckabee, 1973). The maximum values may reflect the industrial activity, but the variability makes definite conclusions impossible. A survey of cadmium in fish from the upper Clark Fork River in western Montana gave the values shown in Table 5.4 (Van Meter, 1974). Mining is common in this part of Montana.

TABLE 5.4. AVERAGE CADMIUM CONCENTRATIONS IN FISH FROM UPPER CLARK FORK RIVER IN WESTERN MONTANA

Species	Cadmium content (ppm wet wt)[a]	
	Muscle	Liver
Trout	0.19	0.69
Whitefish	0.20	0.65
Sucker	0.60	0.79
Sculpin		0.30
Shiner		0.32
Squawfish		0.53

[a] Arithmetic mean of all samples.

Source: Adapted from Van Meter, 1974, Table 2, p. 14.

Cadmium levels in whole fish of three species from the Great Lakes (Lucas, Edgington, and Colby, 1970) averaged 0.094 ppm (range of 0.062 to 0.140 ppm). Concentrations varied among fish of the same species and with collection site. Liver cadmium concentrations in ten other species from the Great Lakes averaged 0.4 ppm (range of 0.06 to 1.4 ppm) and were highest in goldfish from Lake Erie.

Mathis and Cummings (1973) examined levels of cadmium and other metals in organisms from the Illinois River. Results are given in Table 5.5. Bottom sediment contained a mean of 2.0 ppm cadmium (range of 0.02 to 12.1 ppm) and the water a mean of 0.0006 ppm (range of 0.0001 to 0.002 ppm). Bottom-dwelling organisms contained the most cadmium in this study; worms had the highest levels, while fish muscle had the least. There was no significant difference ($P = 0.05$) between muscle cadmium concentrations in carnivorous and omnivorous fish.

Cadmium concentration is much higher in shellfish than in fish. This is true of various kinds of shellfish from around the world (Smith and Huckabee, 1973). The cadmium content of various species of saltwater shellfish from Japan ranged from less than 0.01 ppm in the octopus to 0.85 ppm in the oyster (Smith and Huckabee, 1973).

Seals, although mammals, are discussed in this section because they are aquatic organisms feeding on fish and other seafoods. Cadmium levels in seals from several areas around Britain were determined between 1968 and

TABLE 5.5. CADMIUM LEVELS IN ORGANISMS FROM THE ILLINOIS RIVER

Species	Number of samples	Cadmium level (ppm)	
		Mean	Range
Clams			
Fusconaia flava	17	0.69	0.36-1.17
Amblema plicata	25	0.38	0.15-1.41
Quadrula quadrula	20	0.56	0.31-1.37
Tubificid annelids	11	1.1	0.5-3.2
Carnivorous fish			
Esox lucius	4	0.022	0.013-0.031
Micropterus salmoides	7	0.022	0.004-0.060
Morone chrysops	2	0.024	0.004-0.038
Lepisosteus platostomus	2	0.030	0.004-0.085
Micropterus dolomieui	1	0.005	
Omnivorous fish			
Ictiobus cyprinellus	12	0.032	0.001-0.055
Dorosoma cepedianum	12	0.033	0.005-0.068
Moxostoma macrolepidotum	4	0.017	0.005-0.031
Carpiodes cyprinus	22	0.024	0.004-0.046
Cyprinus carpio	14	0.035	0.011-0.069

Source: Adapted from Mathis and Cummings, 1973, Tables II-V, pp. 1575-1577.

1972 by Heppleston and French (1973). Kidney levels were relatively high (up to 22 ppm wet weight as determined by atomic absorption spectrophotometry) and increased with age as in humans (Section 6). Brain and liver levels were below 1 ppm. Cadmium levels (μg/g kidney wet weight ± standard deviation) were: Farne Island, 5.3 ± 4.0; Outer Hebrides, 11.6 ± 7.5; and Shetland, 2.2 ± 2.0.

5.2.2 Effects

Cadmium produces both acute and chronic effects in aquatic animals; these are considered in this section. Discussion of mechanisms is postponed to Section 6.3.1 because similar molecular mechanisms are presumably involved in all species.

5.2.2.1 Acute — The acute toxicity of cadmium to various aquatic organisms is given in Tables 5.6 and 5.7. Cadmium toxicity varies with concentration, water characteristics, and experimental conditions. Because of these variables, it is difficult to evaluate the relative sensitivities of different

TABLE 5.6. ACUTE DOSES OF CADMIUM FOR SOME AQUATIC ORGANISMS[a]

Species	Acute dose LC_{50}[b] (ppm)	Test conditions
Pimephales promelas	5	Static acute bioassay; hard water; $CdCl_2$
	0.9	Static acute bioassay; soft water; $CdCl_2$
	1.05	Static acute bioassay; soft water; $CdCl_2$, concentration as cadmium
	72.6	Static acute bioassay; hard water; $CdCl_2$, concentration as cadmium
Lepomis macrochirus	1.94	Static acute bioassay; soft water; $CdCl_2$, concentration as cadmium
Lebistes reticulatus	1.27	Static acute bioassay; soft water; $CdCl_2$, concentration as cadmium
Lepomis cyanellus	2.84	Static acute bioassay; soft water; $CdCl_2$, concentration as cadmium
	66.0	Static acute bioassay; hard water; $CdCl_2$, concentration as cadmium
Salmo gairdnerii	0.008-0.01 (7 days)	Continuous flow, acute bioassay; hard water
	30 (1 day)	Continuous flow, acute bioassay; hard water
Crassostrea virginica	0.12 (4-8 weeks)	In flowing water; 20°C; salinity, 31 ppt; $CdCl_2 \cdot 2.5H_2O$
Fundulus heteroclitus	27.0	20-22°C; no feeding during the 96 hr; aerated water

[a] The units used are those presented in the original publication. In some cases it is impossible to deduce whether the concentration is expressed in terms of the element or the compound tested. Very abbreviated descriptions of the conditions of the test are presented. The purpose of the compilation is to indicate the range of concentrations and species used.

[b] Four days, unless specified.

Source: Adapted from National Academy of Sciences and National Academy of Engineering, 1973, Table 1, p. 451. Data collected from several sources.

TABLE 5.7. CADMIUM TOXICITY TO SOME AQUATIC ORGANISMS

Compound	Organism	Field study[a]	Toxicity of active ingredient[b] (ppm)
$CdCl_2$	*Agonus cataphractus*	BSA	33 (T4)
	Cardium edule	BSA	3.3 (T4)
	Crangon crangon	BSA	1.0 (T2)
	Fundulus heteroclitus	L	50 (SB2)
	Fundulus heteroclitus	BSA	55.0 (T4)
	Pagurus longicarpus	BSA	0.32 (T4)
	Palaemonetes vulgaris	BSA	0.42 (T4)
	Asterias forbesi	BSA	0.82 (T4)
	Mya arenaria	BSA	2.2 (T4)
	Carcinus maenus	BSA	4.1 (T4)
	Urosalpinx cinerea	BSA	6.6 (T4)
	Fundulus majalis	BSA	21.0 (T4)
	Mytilus edulis	BSA	25.0 (T4)
	Cyprinodon variegatus	BSA	50.0 (T4)
	Nassarius obsoletus	BSA	10.5 (T4)
	Nereis virens	BSA	11.0 (T4)
$CdSO_4$	*Acroneuria*	BSA	32 (T14)
	Ephemerella	BSA	2 (T4)
	Hydropsyche	BSA	32 (T10)

[a] BSA — bioassay, static, acute; L — laboratory bioassay.

[b] T — TL_m; SB — sublethal effect. The numbers within parentheses following these designations indicate the time in days when the effect was observed.

Source: Adpated from Kemp et al., 1973, Appendix A, pp. A-53 to A-58. Data collected from several sources.

species. Rehwoldt et al. (1971) examined the acute toxicity of cadmium to 12 fish species in soft water (55 ppm as $CaCO_3$). The 96-hr LC_{50} values in these static bioassays varied from 0.11 to 2.8 ppm cadmium. Eisler (1971) reported on the acute toxicity of cadmium to 13 species of marine animals tested at 20°C and 20% salinity. In general, the fish were more resistant, with 96-hr LC_{50} values that ranged from 21 to 51 ppm cadmium. The crustacea were the most sensitive, with 96-hr LC_{50} values ranging from 0.32 to 4.1 ppm cadmium; mollusks and sandworms were intermediate in sensitivity. Biesinger and Christensen (1972) reported a 48-hr LC_{50} value of 0.065 ppm cadmium for *Daphnia magna* tested in Lake Superior waters. Ball (1967) found a one-day LC_{50} value of 30 ppm cadmium; results, as shown in Figure 5.1, suggested a seven-day minimum toxic dose of 0.008 to 0.01 ppm.

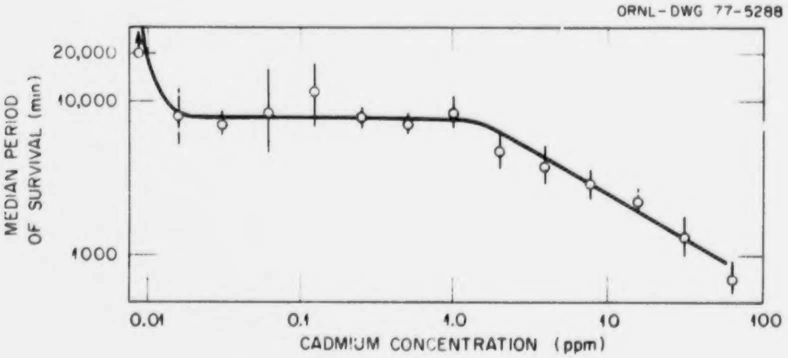


Figure 5.1. Toxicity of cadmium in hard water to rainbow trout. Source: Adapted from Ball, 1967, Figure 1, p. 805. Reprinted by permission of the publisher.

5.2.2.2 Chronic — Chronic toxicity tests are desirable for estimating long-term safe concentrations of cadmium for aquatic life. Pickering and Gast (1972) reported on the chronic toxicity of cadmium in hard water to the fathead minnow. A complete life cycle was studied and the effects on survival, growth, and reproduction were measured. At the higher concentrations mortality slowly increased. The most sensitive effect was on the developing embryos. A cadmium concentration of 0.057 ppm was lethal to some embryos and many surviving larvae were inactive and deformed, whereas 0.037 ppm cadmium did not produce any adverse effects on survival, growth, or reproduction.

Eaton (1974) studied the chronic toxicity of cadmium in hard water to the bluegill. He reported a slow cumulative mortality at all but the lowest cadmium concentration. Continuous exposure in water containing 0.080 ppm cadmium was toxic to bluegills, as judged from effects on larvae and from mortality among adult fish exposed for several months; no effects were produced by cadmium at a concentration of 0.031 ppm.

The chronic toxicity of a mixture of copper, cadmium, and zinc to the fathead minnow was reported by Eaton (1973). He found that the chronic toxicity of the mixture attributable to copper appeared to be increased, but that attributable to cadmium was reduced. The effects thought to be due to zinc were similar to those observed in the single chronic exposure.

Biesinger and Christensen (1972) studied the effects of various metals on survival, growth, reproduction, and metabolism of *Daphnia magna*. Cadmium was the most toxic of the heavy metals. In soft Lake Superior waters they found a three-week LC_{50} value of 0.005 ppm cadmium. A cadmium concentration of 0.0007 ppm depressed reproduction by 50%; a concentration of 0.00017 ppm caused no apparent effects.

5.2.2.3 Mode of Action — Table 5.8 shows some chronic effects of cadmium and suggests some possible modes of action. Gardner and Yevich (1970) exposed the killifish (*Fundulus heteroclitus*) to 50 ppm cadmium in seawater at 37% salinity. They found pathological changes in the intestinal tract as early as 1 hr after exposure, in the kidney after 12 hr, and in the gill filaments and respiratory lamellae after 20 hr. They also found that eosinophils increased to a level 45% above normal.

TABLE 5.8. CHRONIC TOXIC EFFECTS OF CADMIUM TO AQUATIC ORGANISMS

Species	Chronic dose (ppm)	Test conditions
Daphnia magna	0.0026	Threshold of immobilization
Australorbis glabratus	0.05-0.10	Produced distress syndromes; distilled water
Sewage organisms	142	50% inhibition of O_2 utilization
Crassostrea virginica	0.1-0.2	20-week exposure; little shell growth, lost pigmentation of mantle edge; coloration of digestive diverticulae
Fundulus heteroclitus	50	Pathological changes in intestinal tract, kidney, and gills; changes in eosinophil lineage
	27	Inhibition of some liver enzymes

Source: Adapted from National Academy of Sciences and National Academy of Engineering, 1973, Table 2, p. 462.

Jackim, Hamlin, and Sonis (1970) studied the effects of metal exposure on liver enzymes in the killifish (*Fundulus heteroclitus*). Changes in enzyme activity produced by exposure to 27 ppm cadmium in seawater were not necessarily the same in magnitude or direction as those produced in vitro. Fish that survived this cadmium exposure had a significant change in activity of acid phosphatase, xanthine oxidase, and catalase.

Hiltibran (1971) reported on 16 effects of cadmium on oxygen and phosphate metabolism of bluegill liver mitochondria. He found that low levels of cadmium severely interrupt energy production in vitro by blocking oxygen uptake. Oxidation of lactate by the gills of rainbow trout that survived a 24-hr exposure to 1.12 ppm cadmium (50% to 70% survival) was inhibited by over 50% (Bilinski and Jonas, 1973). No detectable effect on oxidation was seen at lower doses although mortality was increased.

5.2.2.4 Alteration of Toxicity — Water conditions such as temperature and salinity might be expected to influence toxicity of cadmium to aquatic organisms, either directly by changing the organism's metabolic rate or indirectly by altering the solubility of the cadmium compound. O'Hara (1973) examined the effect of temperature and salinity on the toxicity of cadmium to the fiddler crab and in general found greater toxicity at higher temperatures and lower salinities. Table 5.9 lists the cadmium level that killed 50% of the crabs (TL_m) for various salinities and temperatures.

TABLE 5.9. CADMIUM TOXICITY TO FIDDLER CRABS AS A FUNCTION OF SALINITY AND TEMPERATURE

Salnity (%)	Time (hr)	TL_m (ppm)[a]		
		10°C	20°C	30°C
10	48			11.0
	96		32.2	6.8
	144	51.0	21.3	4.0
	192	15.7	11.8	2.9
20	48			28.0
	96		46.6	10.4
	144		23.0	5.2
	192	52.0	16.5	3.7
	240	42.0	9.5	3.5
30	48			33.3
	96		37.0	23.3
	144		29.6	7.6
	192		21.0	6.5
	240	47.0	17.9	5.7

[a] Concentration lethal to 50% of crabs.

Source: Adapted from O'Hara, 1973, Table 1, p. 150.

Cadmium toxicity may be altered by the presence of other metals (Section 6.3.2) — a situation likely to exist in most waterways where cadmium is present. With this in mind, Eaton (1973) exposed fathead minnows to mixtures of cadmium, copper, and zinc and compared the toxicities with those of the individual metals. A lethal threshold was reached when each metal was present at a concentration of 40% or less of its individual lethal threshold. The toxicity due to cadmium in the mixture was less than expected from its individual toxicity. Table 5.10 gives acute toxicity levels of the individual metals and of the mixture.

TABLE 5.10. COMPARISON OF THE ACUTE TOXICITY OF A COPPER, CADMIUM, AND ZINC MIXTURE WITH THAT OF INDIVIDUAL METALS TO FATHEAD MINNOWS

Toxicity determination	Measured acute test value (ppb)[a]		
	Copper	Cadmium	Zinc
Copper, cadmium, and zinc mixture, lethal threshold value	145	320	2050
Copper, 96-hr TL_m value[b]	430		
Cadmium, lethal threshold value		7200	
Zinc, lethal threshold value			5030

[a] Mean of daily composited samples.

[b] As little or no mortality took place after three days of exposure, this is considered a close approximation of the lethal threshold value.

Source: Adapted from Eaton, 1973, Table 7, p. 1735. Reprinted by permission of the publisher.

The effect of pollutants on spawning and hatching success is an important consideration in long-term survival of a species in polluted water. Table 5.11 shows the decrease in number of spawnings and number of embryos with increasing concentrations of metals in a mixture of cadmium, copper, and zinc (Eaton, 1973).

5.3 BIRDS

Birds are good indicators of environmental pollution for several reasons. The varied diets of different birds in an area (e.g., seed and grain, rodents and other small animals, or fish) permit a sampling of cadmium contamination of several aspects of the environment. A declining

TABLE 5.11. SPAWNING AND HATCHING SUCCESS OF FATHEAD MINNOWS AT VARIOUS CONCENTRATIONS OF COPPER, CADMIUM, AND ZINC

Concentration (ppb)			Number of females	Number of spawnings	Number of spawnings per female	Total number of embryos produced	Number of embryos per spawning	Number of embryos per female	Number of embryo groups hatched (50 embryos per group)	Embryos hatching[a] (%)
Copper	Cadmium	Zinc								
3.7	0.6 (control)	13.1	14	249	17.8	41,092	165	2935	88	79.4
5.3	3.9	27.3	12	165	13.8	24,403	147	2034	52	78.4
6.7	7.1	42.3	8	27	3.4	4,058	150	507	11	91.8
10.6	14.6	79.4	15	59	3.9	6,224	105	415	16	92.0
17.6	29.5	148.0	15	20	1.3	1,785	89	119	9	86.6
32.0	59.5	302.8	11	3	0.27	51	17	4.6	0;(6)[b]	(93.7)[c]

[a] Over 95% of mortality indicated here occurred before hatching.

[b] The six embryo groups (from three spawnings) in parentheses were transferred from the control tank on the day they were spawned.

[c] Percentage hatch of the embryos transferred from the control tank.

Source: Adapted from Eaton, 1973, Table 4, p. 1730. Reprinted by permission of the publisher.

population trend for a species may also indicate the presence of a toxic substance in the area. In addition to bird muscle, organs, and feathers, eggs can also be analyzed for cadmium.

5.3.1 Metabolism

5.3.1.1 Uptake and Absorption — Birds take up and absorb cadmium via the same routes as other organisms; ingestion contributes the major part of total uptake (Section 6.2.1). Little information is available on actual rates of uptake in birds. Anderson and Van Hook (1973) fed ^{109}Cd-tagged birdseed to chipping sparrows (167 nCi/g birdseed) for 21 days. Cadmium concentrations at 21 days (nCi/g live weight) were: feather, 8; liver, 14; kidneys, 29; and gut, 75. Approximately 8% of the cadmium available in the food was absorbed. Figure 5.2 shows whole-body uptake over the 21-day period. Figure 5.3 shows decrease in whole-body cadmium with time after ^{109}Cd-tagged birdseed was discontinued. After an initial rapid drop in total burden, the remaining cadmium decreased slowly with a half-life of 99 days. The whole-body cadmium level was only about 0.007 ppm live weight; no mention was made of toxic effects.

ORNL-DWG 73-3797

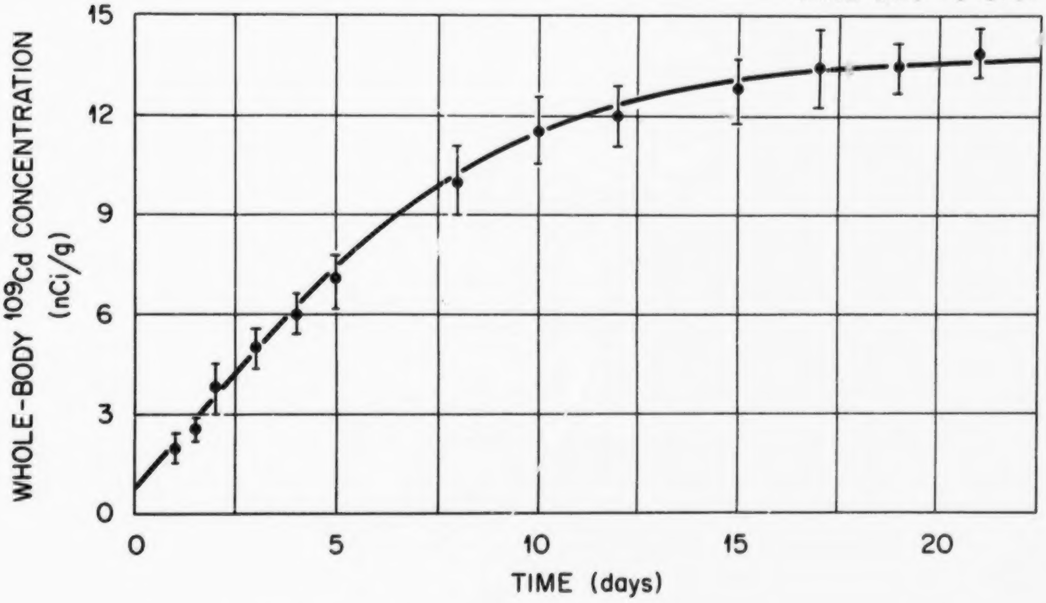


Figure 5.2. Uptake of ^{109}Cd by chipping sparrows feeding on tagged bird seed. Points represent means of 14 observations (± standard error). Source: Anderson and Van Hook, 1973, Figure 1, p. 244.

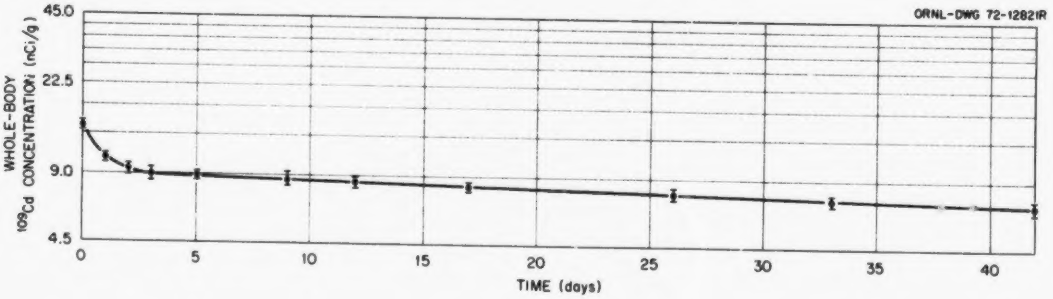


Figure 5.3. Biological turnover of ^{109}Cd by chipping sparrows following 21 days of feeding on tagged bird seed. Points represent means of eight observations (± standard error). Source: Adapted from Anderson and Van Hook, 1973, Figure 2, p. 245.

5.3.1.2 Transport and Distribution — Tissue and organ distribution of cadmium in birds is similar to that in other animals discussed in Section 6.2.2; therefore, transport is probably also similar, although no specific information is available.

Distribution of cadmium in three-month-old female leghorn chickens was determined following 12 weeks of daily intraperitoneal injections of trace amounts of [^{115}Cd] cadmium acetate (average cadmium dose of 0.010 mg/kg body weight) (Dyer, Born, and Kessler, 1974). Three weeks after the last injection radioactivity in tissues was determined. The results are shown in Table 5.12. Because only small amounts of radioactive cadmium had been given, levels are expressed as percentage of whole-body level rather than parts per million of tissue sample. Eggs were laid during the three-week period between the last cadmium injection and sacrifice. On the average, each egg contained approximately 0.1% of the total body cadmium with 80% in yolk, 15% in white, and 5% in shell. The percentage of whole-body cadmium and distribution within the eggs did not change from the first to last egg laid during the three-week period.

5.3.1.3 Normal Levels — Cadmium levels normally found in bird tissues would be expected to vary depending on diet and environmental conditions in a given area. For example, Martin and Nickerson (1973) surveyed cadmium levels in starlings from areas in the United States selected to give a broad coverage of environmental conditions and human activities. Analyses were made on homogenized samples of ten birds (minus skin, beaks, feet, and wings first joint out from body) by atomic absorption spectrophotometry.

TABLE 5.12. PERCENTAGES OF WHOLE-BODY CADMIUM IN CHICKENS AT SACRIFICE

Tissue	Percentage of whole-body cadmium per gram ± std deviation
Liver	0.90 ± 0.33
Kidney	1.88 ± 0.24
Pancreas	0.61 ± 0.21
Spleen	0.30 ± 0.09
Intestines	0.11 ± 0.30
Ceca	0.16 ± 0.07
Stomach	0.15 ± 0.04
Gizzard	0.05 ± 0.02
Shell glands	0.04 ± 0.03
Ovaries	0.03 ± 0.02
Gut fat	0.03 ± 0.00
Carcass	0.02 ± 0.00

Source: Adapted from Dyer, Born, and Kessler, 1974, Table 1, p. 122, by courtesy of Marcel Dekker, Inc.

Values ranged from not detectable (<0.005 ppm) to 0.24 ppm. Seventy-two percent of the samples contained 0.05 ppm or less. Table 5.13 lists the data. It is worth noting that little difference was seen between samples from rural and urban areas.

Cadmium levels in eggs may provide useful information about cadmium levels in birds and their diets. Assuming that the data of Dyer, Born, and Kessler (1974) on cadmium levels in leghorn chicken eggs as percentage of total body burden are applicable to other species (0.1% of total body burden in each egg), it would be possible to quantitate cadmium levels in other species by collecting representative samples of their eggs.

Cadmium levels in the eggs of Cooper's hawks in the Arizona and New Mexico area were determined by Snyder et al. (1973). Cadmium was found in 20 of 24 eggs (limit of detection, 0.015 to 0.024 ppm) with a mean level in those with detectable cadmium of 0.120 ppm (standard error = 0.023 ppm). Overall cadmium levels were quite low; however, levels in eggs from unsuccessful nests were significantly higher than in eggs from successful nests ($P < 0.01$).

A decline in the puffin population in the British Isles led to a search in the birds and their eggs for residues of some organic contaminant or for a particularly high content of heavy metal(s) (Parslow, Jefferies, and French, 1972). Levels of the various pollutants were not unusually high when compared to those in other seabird species with the possible exception

TABLE 5.13. CADMIUM RESIDUES IN STARLINGS IN THE UNITED STATES, 1971

State	City or county	Type of site	Cadmium (ppm, whole-body wet wt)
Alabama	Mobile	Urban	<0.05
Arizona	Phoenix	Rural	0.11
Arkansas	Stuttgart	Rural	0.10
California	Bakersfield	Urban	0.24
	Duplicate analysis		0.11
	Los Angeles	Urban	0.06
	Sacramento	Urban	<0.05
Colorado	Greeley	Rural	0.05
Connecticut	Connecticut River Valley	Rural	<0.05
Delaware	Dover	Rural	0.09
Florida	Gainesville	Rural	<0.05
Georgia	Atlanta	Urban	<0.05
Idaho	Boise	Rural	<0.05
Illinois	Chicago	Urban	0.06
Indiana	Evansville	Rural	
	Highland	Urban	<0.05
Iowa	Des Moines	Urban	ND[a]
Kansas	Garden City	Rural	0.05
Louisiana	Baton Rouge	Rural	<0.05
Maine	Gray	Rural	<0.05
Maryland	Patuxent	Urban	<0.05
	Duplicate analysis		<0.05
Massachusetts	Quincy	Urban	<0.05
Minnesota	Twin Cities	Urban	0.08
Michigan	Lansing	Urban	0.12
Mississippi	Starkville	Rural	<0.05
Missouri	Malden	Rural	<0.05
Nebraska	North Platte	Rural	<0.05

(continued)

TABLE 5.13 (continued)

State	City or county	Type of site	Cadmium (ppm, whole-body wet wt)
Nevada	McGill	Rural	<0.05
	Reno	Rural	<0.05
New Jersey	New Brunswick	Urban	<0.05
	Duplicate analysis		<0.05
New Mexico	Carlsbad	Rural	<0.05
	Farmington	Rural	0.12
	Duplicate analysis		0.10
New York	Albany	Urban	
	Jamestown	Urban	0.08
North Carolina	Raleigh	Urban	<0.05
North Dakota	Mandan	Rural	ND[a]
Ohio	Columbus	Rural	0.08
Oklahoma	Tishomingo	Rural	<0.05
Oregon	Corvallis	Rural	<0.05
	Wilsonville	Rural	0.09
Pennsylvania	Pittsburgh	Urban	<0.05
South Carolina	Columbia	Urban	<0.05
South Dakota	Pierre	Rural	<0.05
	Duplicate analysis		<0.05
Tennessee	Nashville	Urban	<0.05
Texas	Hillsboro	Rural	<0.05
	San Antonio	Urban	<0.05
Utah	Salt Lake City	Rural	0.06
Vermont	Champlain Valley	Rural	<0.05
Virginia	Blacksburg	Rural	<0.05
Washington	Spokane	Rural	<0.05
	Yakima	Rural	<0.05
West Virginia	Elkins	Rural	0.12
Wisconsin	Horicon	Urban	<0.05

[a] ND — not detected.

Source: Adapted from Martin and Nickerson, 1973, Table 1, pp. 69-70.

of cadmium. The cadmium concentrations in the livers of breeding birds from two declining colonies (St. Kilda and Clo Mor) were higher than in livers of puffins from other areas and in livers of other seabirds examined. Table 5.14 shows a comparison of liver cadmium levels in puffins from eight areas. Only 3 of 55 other seabird livers (17 species) had cadmium levels above 12 ppm. No reason for the high cadmium levels in puffins from St. Kilda and Clo Mor was shown.

TABLE 5.14. CONCENTRATIONS OF CADMIUM IN LIVERS OF EIGHT ADULT PUFFINS FROM THE BRITISH ISLES

Locality	Cadmium concentration in liver (mg/g dry wt)	Cadmium concentration in liver Total (μg)
Angus	5.5	8
Aberdeen	6.8	21
Northumberland	3.3	12
	7.5	26
	2.9	10
Farne Islands	8.4	38
St. Kilda	12.9	49
Clo Mor	22.3	127

Source: Adapted from Parslow, Jefferies, and French, 1972, Table VII, p. 28. Reprinted by permission of the publisher.

5.3.2 Effects

Cadmium-induced effects in birds are similar to those produced in other animals as discussed in Section 6.3. In general, the danger to birds is one of chronic exposure to low levels of cadmium, and most work with birds has been with chronic exposures.

Richardson, Spivey Fox, and Fry (1974) fed Japanese quail 75 mg cadmium per kilogram diet from hatching to four or six weeks and compared the effects with those of zinc and iron deficiencies. At four weeks both cadmium-containing and zinc-deficient diets produced growth retardation and testicular hypoplasia. Bone marrow hypoplasia and severe anemia occurred in birds fed cadmium-containing or iron-deficient diets. Hypertrophy of both heart ventricles was apparent at six weeks in cadmium-treated birds. Left-ventricular hypertrophy occurred in four weeks in iron-deficient quail. Ascorbic acid in the diet (1%) prevented cadmium-induced effects.

Similar effects occurred in adult male white leghorn chickens given 2 mg cadmium sulfate per kilogram body weight daily intraperitoneally for 15 to 22 days or a total dose of over 60 mg cadmium per bird (Sturkie, 1973). There was no effect on blood pressure or leucocyte counts, but anemia, enlarged hearts, and myocardial infarction occurred. The hematocrit decreased from an average of 36% before treatment to 19% after 15 cadmium injections. Thirty-four days after the last cadmium injection the hematocrit had increased to 26%, still significantly below normal.

Although cadmium delayed testicular development in the study by Richardson, Spivey Fox, and Fry (1974), testicular damage in birds or other organisms with abdominal testes has not always been reported (Section 6.3.2.4.2; Johnson and Turner, 1972; Johnson and Walker, 1970). Susceptibility differs among avian species. Testicular damage was produced in ring doves 20 days after intramuscular injection of 6.6 mg cadmium per kilogram body weight (Richardson, Spivey Fox, and Fry, 1974), but in domestic pigeons a single subcutaneous injection of 0.5 mg cadmium chloride per kilogram body weight produced damage (Sarkar and Mondal, 1973).

Johnson and Walker (1970) compared the effect of cadmium on carbonic anhydrase activity in rat and domestic fowl testes. Carbonic anhydrase activity was studied because it is a zinc enzyme. Inhibition of carbonic anhydrase activity was similar in both species either in vivo or in vitro; however, testicular damage was apparent only in the rat. Thus, in the rat the inhibition of carbonic anhydrase is not correlated with cadmium damage to the testis. Cadmium increased blood flow in rat but not in domestic fowl testes (Johnson and Turner, 1972); this supports the now generally accepted belief that a primary effect of cadmium is on the testicular vascular system (Richardson, Spivey Fox, and Fry, 1974).

SECTION 5

REFERENCES

1. Anderson, S. H., and R. I. Van Hook, Jr. 1973. Uptake and Biological Turnover of ^{109}Cd in Chipping Sparrows, *Spizella passerina*. Environ. Physiol. Biochem. (Denmark) 3:243-247.

2. Ball, I. R. 1967. The Toxicity of Cadmium to Rainbow Trout (*Salmo gairdnerii* Richardson). Water Res. (Great Britain) 1:805-806.

3. Bilinski, E., and R.E.E. Jonas. 1973. Effects of Cadmium and Copper on the Oxidation of Lactate by Rainbow Trout (*Salmo gairdnerii*) Gills. J. Fish. Res. Board Can. (Canada) 30(10):1553-1558.

4. Biesinger, K. E., and G. M. Christensen. 1972. Effects of Various Metals on Survival, Growth, Reproduction, and Metabolism of *Daphnia magna*. J. Fish. Res. Board Can. (Canada) 29:1691-1700.

5. Cearley, J. E., and R. L. Coleman. 1974. Cadmium Toxicity and Bioconcentration in Largemouth Bass and Bluegill. Bull. Environ. Contam. Toxicol. 11(2):146-151.

6. Dyer, R. D., G. S. Born, and W. V. Kessler. 1974. The Distribution of Intraperitoneally Injected Cadmium-115m in Chickens. Environ. Lett. 7(2):119-124, Marcel Dekker, Inc., N.Y.

7. Eaton, J. G. 1973. Chronic Toxicity of a Copper, Cadmium and Zinc Mixture to the Fathead Minnow (*Pimephales promelas* Rafinesque). Water Res. (Great Britain) 7:1723-1736.

8. Eaton, J. G. 1974. Chronic Cadmium Toxicity to the Bluegill (*Lepomis macrochirus* Rafinesque). Trans. Am. Fish. Soc. 103(4):729-735.

9. Eisler, R. 1971. Cadmium Poisoning in *Fundulus heteroclitus* (Pisces: Cyprinodontidae) and Other Marine Organisms. J. Fish. Res. Board Can. (Canada) 28(9):1225-1234.

10. Eisler, R., G. E. Zaroogian, and R. J. Hennekey. 1972. Cadmium Uptake by Marine Organisms. J. Fish. Res. Board Can. (Canada) 29(9):1367-1369.

11. Gardner, G. R., and P. P. Yevich. 1970. Histological and Hematological Responses of an Estuarine Teleost to Cadmium. J. Fish Res. Board Can. (Canada) 27(12):2185-2196.

12. Heppleston, P. B., and M. C. French. 1973. Mercury and Other Metals in British Seals. Nature (Great Britain) 243:302-304.

13. Hiltibran, R. C. 1971. Effects of Cadmium, Zinc, Manganese, and Calcium on Oxygen and Phosphate Metabolism of Bluegill Liver Mitochondria. J. Water Pollut. Control Fed. 43(5):818-823.

14. Jackim, E., J. M. Hamlin, and S. Sonis. 1970. Effects of Metal Poisoning on Five Liver Enzymes in the Killifish (*Fundulus heteroclitus*). J. Fish. Res. Board Can. (Canada) 27(2):383-390.

15. Johnson, A. D., and P. C. Turner. 1972. Early Actions of Cadmium in the Rat and Domestic Fowl: VI. Testicular and Muscle Blood Flow Changes. Comp. Biochem. Physiol. A (Great Britain) 41:451-456.

16. Johnson, A. D., and G. P. Walker. 1970. Early Actions of Cadmium in the Rat and Domestic Fowl Testis: V. Inhibition of Carbonic Anhydrase. J. Reprod. Fertil. (Great Britain) 23:463-468.

17. Kemp, H. T., R. L. Little, V. L. Holoman, and R. L. Darby. 1973. Water Quality Criteria Data Book, Vol. 5, Effects of Chemicals on Aquatic Life. U.S. Environmental Protection Agency, Washington, D.C. 489 pp.

18. Kumada, H., S. Kimura, M. Yokote, and Y. Matida. 1973. Acute and Chronic Toxicity, Uptake and Retention of Cadmium in Freshwater Organisms. Bull. Freshwater Fish. Res. Lab. (Japan) 22(2):157-165.

19. Lucas, H. F., Jr., D. N. Edgington, and P. J. Colby. 1970. Concentrations of Trace Elements in Great Lakes Fishes. J. Fish. Res. Board Can. (Canada) 27:677-684.

20. Martin, W. E., and P. R. Nickerson. 1973. Mercury, Lead, Cadmium, and Arsenic Residues in Starlings — 1971. Pestic. Monit. J. 7(1):67-72.

21. Mathis, B. J., and T. F. Cummings. 1973. Selected Metals in Sediments, Water, and Biota in the Illinois River. J. Water Pollut. Control Fed. 45(7):1573-1582.

22. Mount, D. I., and C. E. Stephan. 1967. A Method for Detecting Cadmium Poisoning in Fish. J. Wildl. Manage. 31(1):168-172.

23. National Academy of Sciences and National Academy of Engineering. 1973. Water Quality Criteria 1972. U.S. Environmental Protection Agency, Washington, D.C. 594 pp.

24. O'Hara, J. 1973. The Influence of Temperature and Salinity on the Toxicity of Cadmium to the Fiddler Crab, *Uca pugilator*. Nat. Oceanic Atmos. Adm. (U.S.) Fish. Bull. 74(1):149-153.

25. Parslow, J.L.F., D. J. Jefferies, and M. C. French. 1972. Ingested Pollutants in Puffins and Their Eggs. Bird Study (Great Britain) 19(1):18-33.

26. Pickering, Q. H., and M. H. Gast. 1972. Acute and Chronic Toxicity of Cadmium to the Fathead Minnow (*Pimephales promelas*). J. Fish. Res. Board Can. (Canada) 29(8):1099-1106.

27. Rehwoldt, R., L. Menapace, B. Nerrie, and D. Alessandrello. 1971. The Effect of Increased Temperature upon the Acute Toxicity of Some Heavy Metal Ions. Bull. Environ. Contam. Toxicol. 8(2):91-96.

28. Richardson, M. E., M. R. Spivey Fox, and B. E. Fry, Jr. 1974. Pathological Changes Produced in Japanese Quail by Ingestion of Cadmium. J. Nutr. 104(3):323-338.

29. Sarkar, A. K., and R. Mondal. 1973. Injurious Effect of Cadmium on Testis of Domestic Pigeon and Its Prevention by Zinc. Indian J. Exp. Biol. (India) 11:108-109.

30. Smith, R. H., and J. W. Huckabee. 1973. Ecological Studies of the Movement, Fate, and Consequences of Cadmium. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 278-322.

31. Snyder, N.F.R., H. A. Snyder, J. L. Lincer, and R. T. Reynolds. 1973. Organochlorines, Heavy Metals, and the Biology of North American Accipiters. BioScience 23(5):300-305.

32. Sturkie, P. D. 1973. Effects of Cadmium on Electrocardiogram, Blood Pressure, and Hematocrit of Chickens. Avian Dis. 17:106-110.

33. Van Meter, W. P. 1974. Heavy Metal Concentration in Fish Tissue of the Upper Clark Fork River. Montana University Joint Water Resources Research Center, Bozeman, Mont. 37 pp.

SECTION 6

EFFECTS ON TERRESTRIAL MAMMALS INCLUDING MAN

6.1 SUMMARY

This chapter describes effects of cadmium on higher organisms including man. The toxic potential of cadmium, as determined from industrial exposures and animal experiments, is very clear. However, dose-response data for human exposure are scarce. Some estimates of dose levels likely to result in various symptoms and effects in humans are identified in Section 8, along with cadmium concentrations in surface waters, soils, and food.

Human epidemiological data are often deficient in dose information; exposures often include more than one toxic substance, and previous disease and smoking histories may not be known. Controlled animal experiments are therefore essential in providing useful information, in spite of possible problems in extrapolating from animal to man.

In adult humans cadmium is primarily absorbed following inhalation or ingestion; about 25% to 50% of the cadmium retained in the lungs is absorbed, and an average of 6% may be absorbed following ingestion. Once cadmium is absorbed, about 50% is stored in the kidneys and liver. Immediately after an acute exposure most of the cadmium is in the liver, but with time kidney levels increase until one-third of the body burden is located in this organ.

Cadmium is excreted primarily through urine and feces; however, excretion is slow and the half-time of absorbed cadmium in humans is very long — of the order of several decades. The daily dietary intake of cadmium exceeds excretion, leading to an accumulation of cadmium with age. If the concentration reaches about 200 ppm wet weight in the renal cortex, kidney damage may result.

Cadmium reacts with sulfhydryl groups and thus can alter activity of many enzymes, including those involved in energy production. The actual symptoms of cadmium toxicity differ with route and extent of exposure. Acute inhalation exposure causes pulmonary problems. Acute ingestion leads to symptoms mimicking food poisoning and may cause death through shock within 24 hr or from renal failure within one to two weeks. Chronic cadmium inhalation may lead to chronic bronchitis or emphysema after a few years, and maximum permissible exposures have been established in recognition of this risk. The maximum legal atmospheric concentration in the United States for an 8-hr-day exposure period is 0.1 mg/m^3 for cadmium fume and 0.2 mg/m^3 for cadmium dust, although lower limits have recently been recommended. Other effects such as proteinuria, anemia, liver damage, and possibly hypertension may result from long-term cadmium exposure via either inhalation or ingestion. Nutritional factors such as calcium and vitamin D deficiencies can accentuate cadmium effects as may be the case in itai-itai disease in Japan.

The toxicity of cadmium is well recognized, and acute poisoning from inhalation of cadmium fumes or from ingestion of grossly cadmium-contaminated food or drink is rare. The greatest danger lies in long-term, low-level exposure to cadmium, especially in conjunction with low-level exposure to other toxic trace metals in the environment. This chapter emphasizes experimental and epidemiological data concerning uptake and accumulation of cadmium in the body and the actual or possible effects of this accumulation.

6.2 METABOLISM

6.2.1 Uptake and Absorption

6.2.1.1 Inhalation — Cadmium in the air is generally present as an aerosol; therefore, deposition of inhaled cadmium in the respiratory system should be a function of particle size. Absorption of cadmium following deposition is a function of the solubility of the cadmium compound and the rate of clearance. Deposition, retention, and clearance of particulate matter from the respiratory system are discussed in *Air Quality Criteria for Particulate Matter* (U.S. Department of Health, Education, and Welfare, 1969). Figure 6.1 summarizes deposition in the three respiratory compartments with respect to particle size (Hise and Fulkerson, 1973). In general, deposition in the lungs varies inversely with particle size, being roughly 50% for particles with mean mass diameters of 0.1 μ and 10% for particles with mean mass diameters of 5 μ. Deposition in the tracheobronchial system is also an inverse function of particle size, whereas deposition in the nasopharyngeal compartment varies directly with particle size (Figure 6.1). Unfortunately, coal-fired power plants and other high-temperature combustion sources emit the highest concentrations of cadmium in the smallest particles (<1 μ), which are most likely to reach the alveolar region of the lungs (Natusch, Wallace, and Evans, 1974).

Particles deposited in the tracheobronchial compartment should be cleared relatively fast by ciliary activity, while those reaching the lung may have half-times ranging from a few days to a year (Task Group on Lung Dynamics, 1966). Particles cleared from the lung and tracheobronchial system are usually swallowed; larger particles are cleared more rapidly than smaller ones (Hise and Fulkerson, 1973). However, precise data are lacking on clearance of particles from the respiratory system (Task Group on Metal Accumulation, 1973).

Several studies from 1944 to 1947 concerned clearance of cadmium chloride aerosol or cadmium oxide fumes from the lungs of experimental animals. The half-time of an inhaled cadmium chloride aerosol in dog lungs was about five days; however, the particle size was not given (Harrison et al., 1947). In another study, one-fourth of the cadmium chloride initially retained in mice lungs was present at 48 hr (Gerard, 1944, as cited in Harrison et al., 1947). Retention of cadmium oxide fumes in the lungs of mice, guinea pigs, rabbits, dogs, and monkeys was about 11% with a range of 5% to 20%. The widely varying exposure conditions make meaningful conclusions about clearance rates impossible. It is worth noting that cadmium itself, at a concentration as low as 6×10^{-6} *M*, significantly inhibits ciliary activity of hamster trachea in vitro (Adalis et al., 1977). Such inhibition of normal pulmonary clearance mechanisms may be of considerable importance.

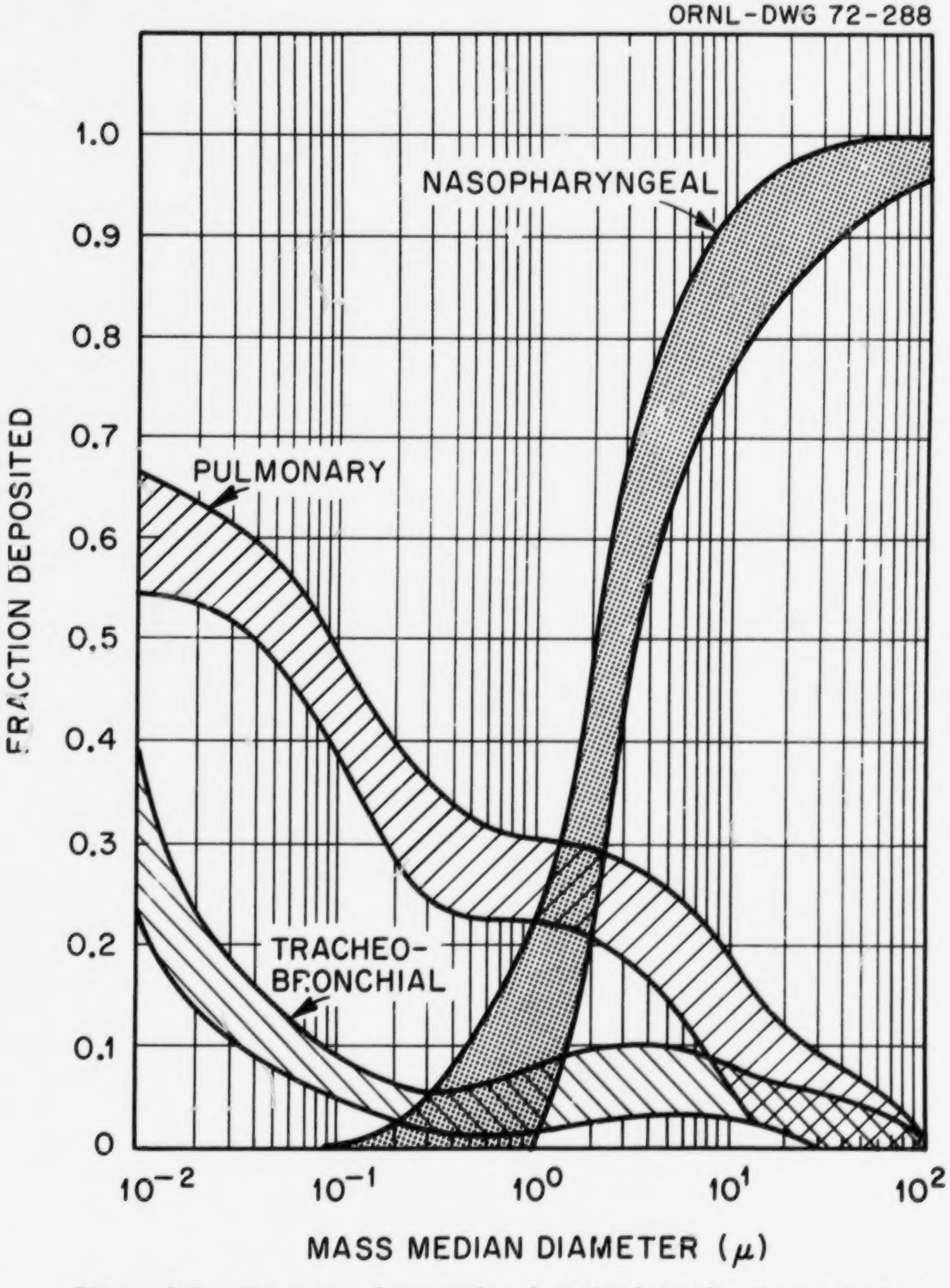


Figure 6.1. Fraction of particles deposited in the three respiratory tract compartments as a function of particle diameter. Source: Hise and Fulkerson, 1973, Figure VI-4, p. 212.

Higher cadmium levels in the organs of persons occupationally exposed to airborne cadmium and in the organs of cigarette smokers [cigarettes contain about 1 to 2 μg of cadmium per cigarette (Section 8.3.2) (Menden et al., 1972)] than in the organs of nonoccupationally exposed, nonsmoking persons demonstrate that absorption of cadmium occurs through the lungs (Adams, Harrison, and Scott, 1969; Barrett and Card, 1947, Bonnell, 1955; Lewis et al., 1972*a*). These epidemiological data do not permit quantitative determinations of absorption rates; however, according to data from smokers, cadmium absorption through the lungs is probably in the range of 25% to 50%, whereas animal experiments have shown an absorption of 10% to 40% of inhaled cadmium (Friberg et al., 1974; Nordberg, 1974).

In general, estimates of body burden and tissue levels have not always distinguished between smokers and nonsmokers. In the population studied by Lewis et al. (1972*b*), total body burden in smokers of around 60 years of age averaged 30 mg; that in nonsmokers averaged only 12 mg.

Because of the undoubted risk of excessive cadmium intake through the lungs, especially in industrial settings, the legal limits for time-weighted average concentrations at the work place in the United States were set at 0.1 mg/m^3 for cadmium fume and at 0.2 mg/m^3 for cadmium dust (Occupational Safety and Health Act, Document 29 CFR 1910). The American Conference of Governmental Industrial Hygienists in 1976 recommended that both levels be reduced to 0.05 mg/m^3; a recent criteria document on cadmium prepared by the National Institute of Occupational Safety and Health (1976) proposed a further lowering to 0.04 mg/m^3. (See also the work of Lauwerys et al., 1974, discussed in Section 6.3.2.3.2.1.)

6.2.1.2 Ingestion — With the exception of occupationally exposed persons or smokers, the cadmium body burden of the general population is acquired mainly through the diet. Most foods and water supplies contain cadmium (Sections 7 and 8). Duggan and Corneliussen (1972) cited an average daily intake of cadmium through foods of 50 μg; this value varies with area of residence and individual choice of food. Cadmium intake from water should be negligible compared with intake from food, except in areas where industrial contamination of the water supply has occurred.

In regard to ingestion of aqueous and solid cadmium, Pribble and Weswig (1973) have suggested the possibility that aqueous cadmium constitutes a different hazard to human health than does dietary cadmium. They observed that weanling brown rats which received 5 mg cadmium (as $CdCl_2$) per liter distilled drinking water accumulated cadmium in the kidneys at a rate 2.5 times that seen in rats receiving 5 mg cadmium (as $CdCl_2$) per kilogram in solid food. For a specified period the rats consumed 1.5 times more water than solid food. The investigators suggested, however, that this does not adequately explain the 2.5-fold difference in accumulation rates between the test groups. A combination of factors, including the physical state of the digestive tract during ingestion, may account for the differential absorption of the metal. Absorption of cadmium from the gastrointestinal tract has been reported as 1% to 75%; however, an average absorption of 6% is probably the most realistic figure (Rahola, Aaran, and Miettinen, 1972).

Tipton, Stewart, and Dickson (1969) and Tipton and Stewart (1969) used atomic absorption measurements to determine the daily cadmium intake in the diet and the daily excretion in urine and feces of three persons for 140 to 347 days. They reported a mean daily intake of 170 μg cadmium, resulting in a fecal excretion rate of 40 μg/day and a urinary excretion rate of 100 μg/day. These values indicated an absorption of 74% of dietary cadmium. The authors apparently were aware of analytical problems referred to by Fulkerson and Goeller (1973) as potentially arising from the presence of high salt concentrations in urine; therefore, there is little reason to suspect that the high cadmium values reported for intake and urinary excretion represent artifacts.

The possible analytical complications in the previous study were, however, completely avoided in a radioactive tracer experiment with five male volunteers aged 19 to 50 years (Rahola, Aaran, and Miettinen, 1972). Each subject received 4.8 to 6.1 μCi of ^{115m}Cd mixed with a calf kidney suspension in a single dose (total ingested cadmium about 100 μg). Average absorption was about 6.0% with a range of 4.7% to 7.0%. Kitamura (1972, as cited in Friberg et al., 1974) reported a gastrointestinal absorption of less than 10% in humans. A low absorption of ingested cadmium (about 2%) has also been found in animal studies (Decker, Byerrum, and Hoppert, 1957; Moore, Stara, and Crocker, 1973). No significant differences were found in absorption of cadmium given to rats as [^{115m}Cd] cadmium sulfate, chloride, or acetate in a single oral dose (Moore, Stara, and Crocker, 1973). The absorption values given above can be modified by dietary factors. For example, low calcium or protein intake doubles the cadmium absorption in animal experiments (Schroeder et al., 1967). The Japanese experience with itai-itai disease (Section 6.3.2.4.7) possibly indicates that dietary factors can also increase cadmium absorption in humans. Ten percent or more of ingested cadmium may be absorbed in humans in the presence of a calcium or protein deficiency (Friberg et al., 1974). Vitamin D was also shown to increase cadmium uptake into the tibia of rachitic chickens when cadmium was given orally but not when it was injected (Worker and Migicovsky, 1961), indicating an effect on intestinal absorption. Absorption can vary with the particular cadmium salt ingested. Twice as much cadmium was found in the livers of rats given cadmium sulfate as in rats given comparable amounts of cadmium as cadmium stearate (Friberg et al., 1974).

Miller et al. (1967) gave three holstein cows 3.0 g each of cadmium as $CdCl_2$ daily in two equal doses in gelatin capsules for two weeks. During the second week, an average of 82% of the cadmium given to the cows was excreted in the feces. Cadmium in the urine was below the limits of detection — 0.5 ppm of urine by atomic absorption spectrometry. Assuming that the fecal samples taken were representative of total feces, cadmium absorption over the two-week period was equivalent to about 12 ppm of the cow's weight. Excretion through milk was very low, and all samples collected during and after cadmium administration had a cadmium level less than 0.1 ppm of milk, the limit of detection. Total cadmium excreted in milk was less than 0.022% of administered cadmium, possibly up to ten times less (Section 6.2.3.4).

Miller, Blackmon, and Martin (1968) gave 2.5-month-old goats single tracer doses of ^{109}Cd as $CdCl_2$, either orally or intravenously. About 90% of the oral dose was excreted in the feces within five days, a value comparable to that reported in the previous study for cows and similar to those for other mammals. Following intravenous administration, however, only 5.6% of the dose was excreted in the feces in five days. Only significant fractions of the cadmium administered either way were excreted in the first seven days.

6.2.1.3 Placental Transfer — The effectiveness of the placental barrier in preventing cadmium from reaching the fetus apparently varies with the dose and route of administration (Friberg et al., 1974). Thus, when cadmium is administered via routes that provide rapid and quantitative absorption (i.e., intravenously or intraperitoneally) and at relatively high dosage rates — 0.5 to 2.0 mg/kg and above — significant fetal cadmium accumulations or teratogenic effects are observed (Barr, 1973; Ferm and Carpenter, 1968; Mulvihill, Gamm, and Ferm, 1970; Tanaka et al., 1972) (Section 6.3.2.4.5). In contrast, cadmium in small doses, even when given intravenously, was not observed to accumulate in fetal tissues of mice (Berlin and Ullberg, 1963).

Experiments reported in preliminary form (Choudhury et al., 1977) showed that cadmium given to pregnant rats in drinking water caused significant biochemical and behavioral changes in the offspring. The oral administration of cadmium to the mothers in these studies did not add significant amounts of the metal to the body burden of the neonates.

Large doses of cadmium (2.5 to 12 mg/kg) administered subcutaneously at various times during gestation in rats have generally caused fetal anomalies or deaths (Barr, 1973; Chernoff, 1973; Parizek, 1964, 1965). Fetal cadmium levels were not reported in the above studies although destruction of the placenta occurred at 2.5 mg cadmium per kilogram (Friberg et al., 1974; Parizek, 1965).

Cadmium levels in fetal tissues have been studied by Ishizu et al. (1973). These workers injected a single subcutaneous dose of cadmium (2.5 mg/kg as cadmium chloride) on day 7 of gestation; fetuses were taken on day 18. The dose was sufficient to cause teratogenic effects. Although cadmium concentrations in the placentas of treated mice increased tenfold, similar fetal cadmium levels were observed for both groups. From these results the authors concluded that placental transfer of cadmium per se might not be necessary to produce adverse fetal effects. Rather, they suggested that the cadmium effect might be exerted on the placenta. However, because cadmium levels in the fetuses were only measured approximately 11 days after a single injection, the results of these studies do not exclude the possibility of significant placental transfer of cadmium and, thus, a direct effect on the fetus. This possibility is further reinforced by the observation of Ferm, Hanlon, and Urban (1969), who studied the distribution of cadmium in fetal tissues 24 and 96 hr after a single injection of the metal into pregnant hamsters; levels after 96 hr were significantly lower than those observed after 24 hr.

These experimental results indicate that the placenta is not a complete barrier against cadmium. As pointed out by Chernoff (1973), the placenta may block the transfer of small amounts of maternally administered cadmium, but larger doses will enter the fetus. For example, Barr (1973) found that a dose of cadmium capable of inducing teratogenesis when given intraperitoneally was not able to induce anomalies when given subcutaneously. Measurements of cadmium in human fetal tissues support the observation of Chernoff (1973). Indeed, in areas where maternal cadmium intake is considered greater than "normal," the cadmium body burden of fetuses will generally also be higher (Chaube, Nishimura, and Swinyard, 1973; Henke et al., 1970, as cited in Friberg et al., 1974).

6.2.1.4 Other Routes of Uptake — Cadmium in solution has been shown to penetrate guinea pig skin (Skog and Wahlberg, 1964) as well as rabbit and mouse skin (Kimura and Otaki, 1972); presumably it also would penetrate human skin in the unlikely case of cutaneous exposure. Various types of injection (subcutaneous, intraperitoneal, intravenous, and intramuscular) are only of interest from an experimental viewpoint. Moore et al. (1973) compared the initial absorption of cadmium administered by different routes to rats: 93% for intraperitoneal injection, 91% for intravenous injection, 41% for inhalation, and 2.3% for ingestion.

6.2.2 Transport and Distribution

6.2.2.1 In Blood — Whether absorbed from the respiratory or gastrointestinal tract, cadmium is distributed in and transported by the blood. Experimental data on initial distribution and transport in the blood are derived from animal studies. Blood values determined in humans reflect a long-term balance since exposure continues from birth. Acute exposure potentially provides more meaningful data on early blood distribution, but acute exposures usually occur with persons exposed occupationally to higher cadmium levels than is the general population, thus complicating the interpretation. The ratio of cadmium in plasma to that in cells of persons with no known exposure to cadmium, except that normally acquired through diet and air, was 1.9 in a study by Szadkowski (1972, cited in Friberg et al., 1974). The mean blood cadmium level in this study of 18 individuals was 3.5 ng/g, as determined by atomic absorption spectrophotometry after extraction. A wide variation in "normal" blood levels appears in the literature (Section 6.2.5); however, an average normal level in whole blood or serum is probably well below 10 ng/g. Chronically exposed workers have higher blood cadmium levels than normal workers (Sections 6.2.5.1 and 6.2.6), and more cadmium is found in cells than in plasma. A mean plasma-to-cell cadmium distribution ratio of 0.5 was reported for 22 exposed workers (Friberg et al., 1974). These observations are in agreement with results determined experimentally in animals.

Several studies using dogs, rabbits, or rats show that during the first hours after intravenous injection of cadmium, blood cadmium is found mainly in the plasma — 98% at 0.5 min and 99% at 8.5 min after injection in rats (Perry et al., 1970) and 90% to 80% at 9 min and at 1 hr after injection in rabbits (Kench et al., 1962, as cited in Friberg et al., 1974). Clearance from the blood is initially quite fast during the first 10 min after injection but is followed by a slower clearance phase (Walsh and Burch, 1959). Intraperitoneal and subcutaneous injections gave similar results (Friberg et al., 1974).

Cadmium concentrations in red cells and plasma of mice were monitored following subcutaneous injection of 1 mg/kg $^{109}CdCl_2$ (Nordberg, 1972). Results are shown in Figure 6.2. Red cells were hemolyzed and fractionated on G-75 Sephadex to determine cadmium distribution. Twenty minutes after injection most of the cadmium was in a high-molecular-weight fraction with only a minor amount in the hemoglobin fraction and essentially none in the metallothionein fraction. Up to 4 hr after injection most cadmium in plasma was in fractions corresponding to albumin or larger proteins.

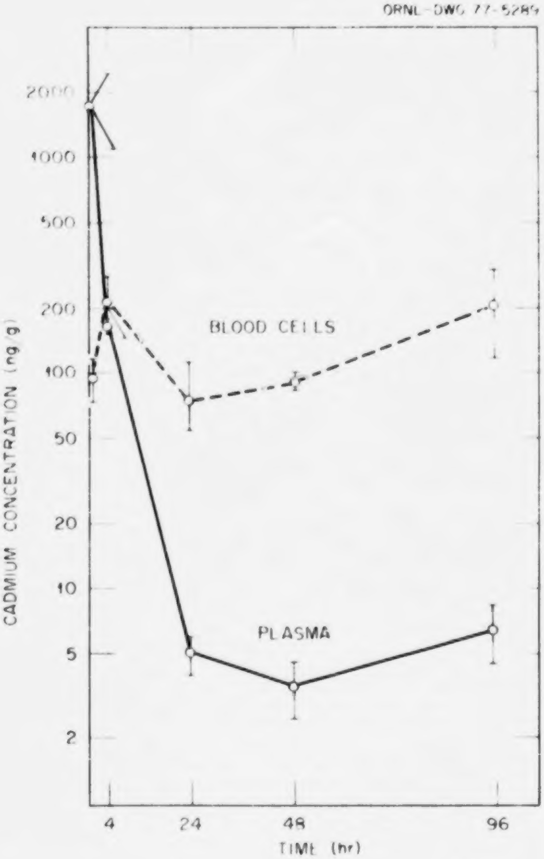


Figure 6.2. Concentrations of cadmium in plasma and blood cells in mice given a single subcutaneous injection of $^{109}CdCl_2$ and killed various times after injection. Vertical bars indicate ranges of two or three mice and circles are mean values. Source: Adapted from Nordberg, 1972, Figure 1, p. 11. Reprinted by permission of the publisher.

Twenty-four hours after injection, cadmium in blood is found mainly in the cells and appears in a low-molecular-weight protein fraction, probably metallothionein (Friberg et al., 1974). Insufficient metallothionein is probably available to handle acutely injected cadmium; metallothionein will therefore be involved only to a minor degree in transport of cadmium during the first hours after injection (Friberg et al., 1974). Nevertheless, Friberg et al. (1974) concluded that proteins, such as metallothionein, hemoglobin, and others, may play important roles in cadmium metabolism.

Following initial clearance of cadmium from the blood, a subsequent increase in blood cadmium levels has been seen in animals. For example, the blood cadmium concentration in rats 16 days after an intravenous injection of [^{115}Cd] cadmium sulfate was twice the level at one day (Niemeir, 1967, as cited in Friberg et al., 1974). Increases in blood cadmium after an initial decrease were also shown in goats and hamsters (Ferm, Hanlon, and Urban, 1969; Miller et al., 1967). This increase probably reflects a release of cadmium from the liver into the blood.

Distribution of cadmium in the blood of animals receiving repeated cadmium injections is similar to that seen in exposed workers. More cadmium appears in the cells than in the plasma. Rabbits given subcutaneous injections of [^{115}Cd] cadmium sulfate at 0.65 mg/kg six days a week for four or ten weeks had blood cadmium levels of 450 and 1000 ng/g respectively (Friberg, 1952; Friberg et al., 1974). Cadmium was not detected in the plasma. Similar results were obtained when rabbits were subcutaneously given 13 injections of 2.1 mg/kg cadmium sulfate or 19 injections of 1.8 mg/kg cadmium sulfate. The red blood cells contained 18 and 10 times more cadmium, respectively, than the plasma (Truhaut and Boudene, 1954, as cited in Friberg et al., 1974).

The concentration of cadmium in blood has been shown to decrease in workers after exposure stops (Rogenfelt, personal communication cited in Friberg et al., 1974). The half-time in blood was about six months. These observations have been confirmed in animal studies.

6.2.2.2 Organ Distribution — Many studies on cadmium distribution in organs of various groups around the world have been published — for example, reviews by Friberg et al. (1974) and Nordberg (1972) and a report by Forssen (1972). Actual values in the organs vary, but the relative distribution among organs is quite similar. Kidneys and liver accumulate far more cadmium than any other organs. Over half of the total body burden may be found in these two organs, with perhaps one-third in the renal cortex.

Table 6.1 lists cadmium levels in 43 different organs taken at autopsy from males and females who had lived their entire lives in Finland. Ages ranged from birth to 70 years; death resulted from causes other than disease. Only 20 subjects were involved and cadmium levels were not determined in all organs of all subjects; however, relative distribution agrees with that obtained in other studies. The ratio of cadmium concentration in kidney to that in liver in this Finnish study was about 10. In the United States, mean ratios between 11 and 15 were found for individuals between ages 20 and 59 grouped in ten-year age intervals (Schroeder and Balassa, 1961),

TABLE 6.1. CADMIUM LEVELS IN ORGANS OF FINNISH MALES AND FEMALES AT AUTOPSY[a]

Organ	Number of samples	Number of positive findings	Cadmium concentration (% in ash)[b]			Ash (% of dry wt)[c]
			Median	Min	Max	
Parietal lobe						
Gray matter	20	0	<	<	<	7.08
White matter	20	1	<	<	0.002	6.35
Hypothalamus	16	0	<	<	<	6.73
Aorta	18	0	<	<	<	2.79
Myocardium	20	1	<	<	0.002	5.03
Vena cava	14	2	<	<	0.004	3.46
Larynx	19	0	<	<	<	12.02
Trachea	18	1	<	<	0.004	4.88
Lung						
Upper lobe	19	10	0.002	<	0.017	5.78
Middle lobe	19	10	0.002	<	0.009	5.70
Lower lobe	18	11	0.002	<	0.014	5.22
Esophagus	18	1	<	<	0.003	4.27
Stomach	18	3	<	<	0.005	4.52
Duodenum	18	7	<	<	0.006	4.78
Jejunum	18	5	<	<	0.013	4.78
Ileum	17	4	<	<	0.004	4.75
Cecum	18	4	<	<	0.005	3.62
Sigmoid colon	18	2	<	<	0.003	3.86
Rectum	18	2	<	<	0.005	2.84
Pancreas	19	12	0.002	<	0.009	4.25
Liver	20	14	0.007	<	0.019	5.40
Gall bladder	12	4	<	<	0.005	4.62
Kidney	19	17	0.072	<	0.281	5.66
Urinary bladder	18	6	<	<	0.006	2.79
Testis	12	3	<	<	0.006	6.57
Prostate	12	1	<	<	0.007	4.74
Ovary	5	2	<	<	0.003	5.75
Uterus	5	0	<	<	<	5.35
Vagina	5	0	<	<	<	4.86
Breast	4	0	<	<	<	0.98
Adrenal	16	11	0.002	<	0.006	2.77
Thyroid	17	12	0.003	<	0.017	4.16
Spleen	20	1	<	<	0.003	5.60
Lymph nodes	6	0	<	<	<	3.16
Thymus	7	0	<	<	<	6.09
Bone marrow	20	1	<	<	0.007	27.72

(continued)

TABLE 6.1 (continued)

Organ	Number of samples	Number of positive findings	Cadmium concentration (% in ash)[b]			Ash (% of dry wt)[c]
			Median	Min	Max	
Skeletal muscle	19	1	<	<	0.004	4.03
Articular cartilage (knee)	19	0	<	<	<	27.98
Costal cartilage	19	0	<	<	<	25.98
Bone (rib)	20	0	<	<	<	33.76
Skin (mid-ventral)	13	0	<	<	<	1.15
Fat (under skin mid-ventral)	8	3	<	<	0.004	0.22
Hair	6	2	<	<	0.003	1.36

[a] Samples dry ashed in quartz crucibles at 200°C for some hours, then at 450°C overnight. Lowest positive value used was 0.002% in ash.
[b] < — value below detection limit.
[c] Median.

Source: Adapted from Forssen, 1972, Table 3, pp. 105-126. Reprinted by permission of the publisher.

whereas seven groups, each with a mean age of 60 years, had ratios between 9 and 14 (Morgan, unpublished data cited in Friberg et al., 1974).

Cadmium distribution between liver and kidney varies in some circumstances. During or immediately after exposure, the liver may contain as much or more cadmium than the kidneys, which could explain why occupationally exposed workers usually have lower kidney-to-liver cadmium ratios than unexposed workers (Friberg et al., 1974).

Rats given intravenous injections of [^{115}Cd] cadmium nitrate (0.65 mg/kg) had 62% to 70% of the injected dose in liver and only 1.6% to 2.4% in kidneys at 4 hr to five weeks after injection (Decker, Byerrum, and Hoppert, 1957). Similar results were obtained following intravenous injection of [^{115}Cd] cadmium nitrate (0.2 to 0.4 mg/kg) into dogs. The liver concentration was much greater than the kidney concentration during the first 24 hr, but after three to four weeks the kidney levels were 50% to 100% of the liver levels (Burch and Walsh, 1959).

The route of administration can have an effect on cadmium distribution in tissues. Table 6.2 shows tissue distribution of cadmium two weeks after oral or intravenous administration into goats. Cadmium may be transported in blood in a different form when absorbed from the digestive tract than when injected, and it may also be metabolized differently.

TABLE 6.2. TISSUE DISTRIBUTION OF ^{109}Cd 14 DAYS AFTER ORAL OR INTRAVENOUS ADMINISTRATION TO GOATS[a]

Tissue sampled	Cadmium concentration (% of dose per kg)	
	Oral dose	Intravenous dose
Hair	0.11	1.9
Skin	0.02	1.3
Testicles	0.02	2.3
Tibia		
Joint	0.07	1.9
Shaft	0.04	0.9
Kidney	1.13	49.2
Muscle	0.03	0.6
Liver	0.88	129.9
Heart	0.02	3.0
Spleen	0.05	17.2
Duodenum[b]	1.04	4.3

[a] All tissue data expressed on a fresh weight basis.

[b] First 1.83 m of small intestine.

Source: Adapted from Miller, Blackmon, and Martin, 1968, Table 1, p. 1838. Reprinted by permission of the publisher.

Miller et al. (1969) determined the effect of added stable cadmium in the diet on the distribution of trace amounts of ^{109}Cd in goats. Four- to six-month-old male goats were fed 100 ppm cadmium as $CdCl_2$ in the diet starting seven days before a single oral tracer dose of ^{109}Cd and continuing until sacrifice 14 days after the tracer dose. This level of cadmium was near the upper limit before toxic symptoms appeared. Total body retention of the tracer dose was 0.3% to 0.4% and, with the exception of reduced retention of ^{109}Cd in some sections of the gastrointestinal tract, there was no significant difference in tissue distribution between goats receiving 100 ppm stable cadmium and those that did not. The rate of daily excretion following absorption also remained unaffected, decreasing in both experimental and control goats during the second week after dosing from 20% of body burden on day 8 to less than 5% of remaining body burden on day 14.

6.2.3 Excretion

Cadmium is excreted primarily in urine and feces, with minor amounts potentially excreted in hair, sweat, skin, milk, and saliva (Friberg et al., 1974; Hise and Fulkerson, 1973).

6.2.3.1 Urinary Excretion — Urinary excretion of cadmium varies; however, an average value of 1 to 2 μg/day is commonly reported for normal, unexposed individuals (Table 6.3). No, or only a slight, relationship has been found between age and urinary excretion of cadmium (Suzuki and Taguchi, 1970; Szadkowski, Schaller, and Lehnert, 1969). Another study, however, showed a rise in urinary excretion (0.5 to 2 μg/liter) from age 5 to 35 with a leveling off until age 55, when excretion slowly fell (Friberg et al., 1974; Nordberg, 1972; Tsuchiya, 1967). The youngest subject in the study of Suzuki and Taguchi (1970) was 16 years old, possibly masking the relationship. Despite a large individual scatter, Nordberg (1972) found an increase in urinary cadmium with total body burden on a group basis in mice; total body burden increases with age. Table 6.3 lists the urinary excretion of cadmium from normal, unexposed individuals.

TABLE 6.3. CADMIUM EXCRETION IN URINE OF NORMAL, UNEXPOSED INDIVIDUALS

Country	Analytical method	Number of individuals	Age range (years)	Cadmium level (μg/liter)		
				Mean	Standard deviation	Range
United States	Spectrographic after dithizore extraction	154		1.59		<0.5-10.8
Japan (Gifu)	Atomic absorption after extraction	46	4-6	0.47	0.25	
		40	9-10	0.65	0.45	
		41	14-15	0.72	0.50	
		56	20-29	0.99	0.63	
		37	30-39	1.13	1.06	
		40	40-49	1.76	1.33	
		43	50-59	1.75	1.38	
West Germany	Dithizone	14		1.0		
Sweden	Atomic absorption after extraction with chelating agent into organic solvent	10[a]	34-63	2.1		
		10	20-40	0.34		

[a] Polluted area.

Source: Adapted from Friberg et al., Cadmium in the Environment, 2nd ed., Table 4:7, pp. 66-67, (c) CRC Press, Inc., 1974. Used by permission of CRC Press, Inc.

There is no clear relationship between urinary cadmium excretion in exposed workers and degree of exposure. In exposed workers, urinary excretion of cadmium varies from 0 to 1000 μg/day (Adams, Harrison, and Scott, 1969; Truhaut and Boudene, 1954, as cited in Friberg et al., 1974). Workers who have absorbed "enough" cadmium (defined in Section 6.3.2.3.2.1) to produce kidney damage and proteinuria have higher cadmium levels in urine than exposed persons without proteinuria. Some workers with proteinuria studied by Adams, Harrison, and Scott (1969) excreted 30 to 170 μg/liter cadmium in their urine, whereas other workers in the same study, without proteinuria, had less than 2 μg/liter.

Urinary excretion of cadmium usually decreases after exposure stops, but not in a regular, predictable pattern. Unpublished data from Piscator (cited in Friberg et al., 1974) showed large individual variations in urinary cadmium in workers one or two years after cadmium exposure ceased. Some workers showed an increase, some a decrease, and some no change.

6.2.3.2 Fecal Excretion — The amount of cadmium excreted by the intestines cannot be determined from fecal cadmium levels since unabsorbed cadmium from the diet passing through the digestive tract will add to any cadmium actually being excreted by the intestines. Therefore, fecal cadmium levels reported for humans give no information about intestinal excretion. Fecal excretion of cadmium in normal, unexposed persons averaged 31 μg/day in a study by Essing et al. (1969) and 42 μg/day in another study by Tipton and Stewart (1969).

Less than 0.1% of the retained oral dose of ^{115m}Cd in humans was excreted in feces (Rahola, Aaran, and Miettinen, 1972). The mechanisms of fecal cadmium excretion in humans are not known. High concentrations of cadmium in bile from autopsy samples indicate an enterohepatic circulation of cadmium (Tsuchiua et al., 1972, as cited in Friberg et al., 1974). However, experiments with rats given [^{109}Cd] cadmium sulfate intraperitoneally or [^{109}Cd] cadmium chloride subcutaneously, while indicating excretion via bile with some recirculation, showed that only about 5% of the fecal excretion could be accounted for by this route (Caujolle, Oustrin, and Silve-Mamy, 1971; Nordberg and Robert, unpublished data cited in Friberg et al., 1974).

Nordberg (cited in Friberg et al., 1974) exposed mice to 0.025 or 0.25 mg cadmium per kilogram body weight five days a week for five months and found that the main part of fecal excretion of cadmium was correlated with daily dose. However, since the average fecal cadmium was higher after exposure stopped than before exposure began (14.2 μg/24 hr, standard deviation ±11, versus 5.1 ± 3.1 μg), part of the fecal excretion must be related to body burden.

6.2.3.3 Excretion in Hair — Schroeder and Nason (1969) examined cadmium levels in hair as a function of age, sex, and hair color. Results showed some differences in cadmium content with color (males had less cadmium in black hair than other colors, and women had less cadmium in gray than other colors), with age, and with sex (female gray hair had less cadmium than male gray hair). Concentration of cadmium in hair probably does not reflect tissue stores under normal conditions (Schroeder and Nason, 1969).

Hammer et al. (1971) found a positive correlation in ten-year-old boys between cadmium levels in hair and community exposure to cadmium. Boys from five cities ranked according to trace-metal contamination in the area were used as subjects (Table 6.4).

TABLE 6.4. CADMIUM IN HAIR FROM BOYS LIVING IN URBAN AREAS

Exposure ranking of city[a]	Number of determinations	Cadmium content of hair[b] (ppm)
I. High	45	2.1
II. High	25	1.5
III. Low[c]	37	1.0
IV. Low	21	1.0
V. Low	37	0.7

[a] Ranking determined by combining aerometric, geologic, and industrial data.

[b] Geometric mean.

[c] Low approximates usual U.S. urban level.

Source: Adapted from Hammer et al., 1971, Table 3, p. 86. Reprinted by permission of the publisher.

More recent studies cited by Friberg et al. (1975) indicate that levels of cadmium in hair in the United States range from 1 to 2 ppm, varying in part with age and sex of the individual. Lower values have been reported from Europe and Japan; European values averaged about 0.5 ppm. No obvious differences in analytical methodology or in exposure to cadmium as reflected in levels of the metal in liver and kidney can account for this discrepancy. The usefulness of cadmium levels in hair as an index of total body burden of cadmium remains, therefore, in question.

6.2.3.4 Excretion in Milk — In terms of the clearance of cadmium from the body, only the fecal and urinary routes appear to be of significance. Because excretion into milk, however, may be a major source of dietary cadmium, it deserves special mention. Indeed, according to Pinkerton et al. (1973), excretion in human or bovine milk can contribute 25% to 50% of the total human dietary uptake in the first years of life.

The studies of Miller discussed in Section 6.2.2.2 have shown that absorption of cadmium following ingestion is relatively low in ruminants and that only a small fraction of this cadmium is excreted in milk. Cadmium levels in cow milk from cities around the United States were found to average between 0.017 and 0.030 ppm as determined by atomic absorption spectrophotometry (Murthy and Rhea, 1968). The national weighted average was 0.026 ± 0.004 ppm. Although low, this value is more than twice the level established for cadmium in drinking water (U.S. Department of Health, Education, and Welfare, 1962). These values, if accurate, could be of concern to those who ingest large quantities of milk. A person consuming 1 qt of milk daily would ingest 30% to 50% of what is considered the average daily intake of cadmium from this one source (Section 6.2.1.2). In spite of the importance of extraction in the analysis for cadmium (Friberg et al., 1974), adequate control experiments have shown no reason to assume that the above data on milk are too high (Murthy, 1974). More recently, somewhat lower values were reported by Bruhn and Franke (1974) using extraction procedures on 315 samples of California milk; an average concentration of 0.0060 ppm (standard deviation, 0.0040 ppm) was found.

A study in the United Kingdom by the Ministry of Agriculture, Fisheries, and Food (1973), based on extraction and atomic absorption techniques, reported a mean concentration for all milk samples of less than 0.002 ppm, a value ten times less than that reported by Murthy and Rhea (1968). Cornell and Pallansch (1973) used pulse polarography with internal standards and found natural concentrations of cadmium in dried, skimmed cow milk of 0.7 to 2.9 ppb. On a fresh milk basis, this value would correspond to a level 100 times lower than that observed by Murthy and Rhea (1968).

6.2.4 Biological Half-Time

The biological half-time of absorbed cadmium in the body is very long and, as a result, accumulation is essentially continuous throughout life. Newborn babies, for example, have an average cadmium concentration of about 0.002 ppm in liver and 0.010 ppm in kidney, whereas values in 50-year-old men averaged about 3 ppm in liver and 30 to 50 ppm in renal cortex (Nordberg, 1974).

An exact biological half-time for cadmium in the human body cannot be determined from available experimental data. Rahola, Aaran, and Miettinen (1972) gave ^{115m}Cd orally (4.8 to 6.1 μCi/100 g in calf kidney suspension) to five men. After an initial rapid decrease in whole-body retention during the first week, probably representing unabsorbed cadmium, a slower elimination phase was seen. The slow decrease in whole-body retention was only followed for two months, and the estimated half-time of this slow phase could be anywhere from 130 days to infinity.

An estimate of cadmium half-life in the human body can be made on a group basis by comparing body burden and excretion. Based on data from various countries, a half-time of 13 to 47 years was obtained by Friberg et al. (1974). Similar biological half-times were calculated on theoretical models. All human data favor a very long biological half-time.

Figure 6.3 shows whole-body retention of ^{115m}Cd in rats following a single exposure by different routes. Except for differences in the initial rapid decay, route of administration did not significantly influence the rate of elimination and biological half-life (Moore et al., 1973).

The toxicological significance of experimentally established values for the biological half-time of cadmium is thrown into doubt by the facts

ORNL-DWG 77-5290

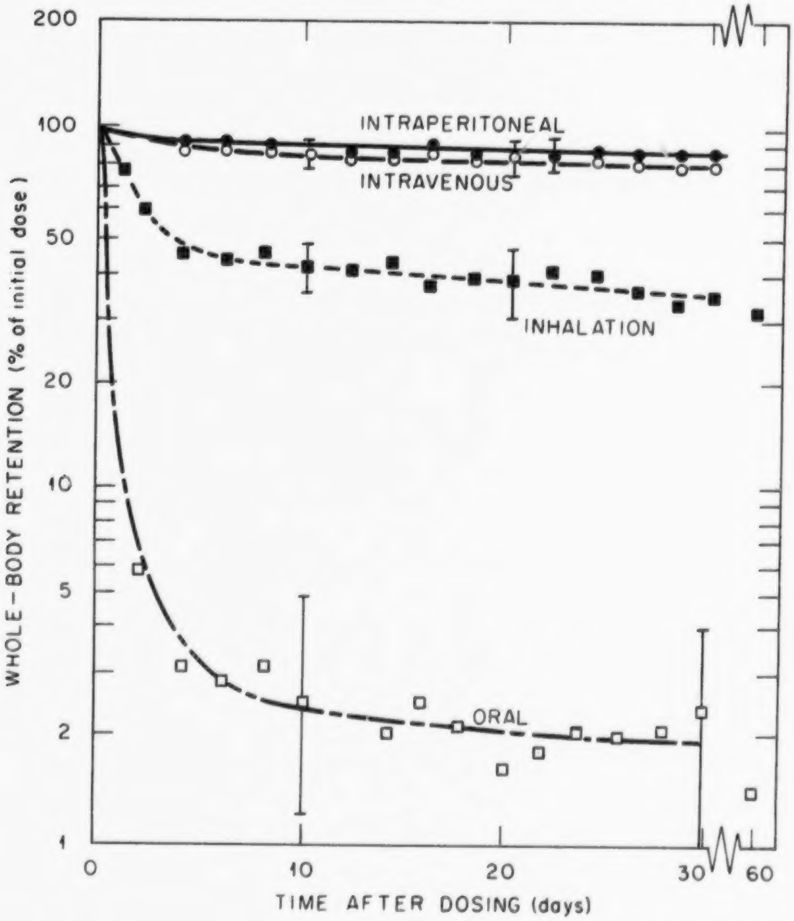


Figure 6.3. Whole-body retention of ^{115m}Cd in rats following a single exposure by different routes of administration. Source: Adapted from Moore et al., 1973, Figure 1, p. 475. Reprinted by permission of the publisher.

that (1) different half-lives for cadmium have been observed in different tissues, and even within one tissue the chemical form of the retained metal is known to change with time, and (2) the half-life is affected by the total dose of cadmium administered and by environmental factors such as temperature. For a fuller discussion of these points see Nomiyama et al. (1977).

6.2.5 Normal Levels

6.2.5.1 In Blood — As mentioned in Section 6.2.2.1, the average "normal" cadmium level in human whole blood or serum lies below 0.01 ppm. Table 6.5 lists some representative values in whole blood of normal unexposed persons

TABLE 6.5. CADMIUM CONCENTRATION IN WHOLE BLOOD FROM "NORMAL" PERSONS AND EXPOSED WORKERS

Country	Group	Number of individuals	Cadmium in whole blood (ppb)			Method of analysis
			Mean	Median	Range	
Sweden	Normal	6	5		2-8	Neutron activation
	Normal	150	<2	<2		Spectrographic
West Germany	Normal	18	3.5			Atomic absorption after extraction
Belgium	Normal	24	9.5			Atomic absorption after extraction
United States	Normal	153	8.5	7.0	3.4-53.5	Spectrographic after extraction
	Normal	243	<5	<5	<5-142	Atomic absorption after extraction
	Normal	50		0.6	<0.2-2	Atomic absorption (delves cup technique)
United Kingdom	Normal children	89	4.9		0-19	Atomic absorption after extraction
	Hospitalized children	204	5.7		0-79	Atomic absorption after extraction
Sweden	Presently exposed workers	22	27		4-58	Spectrographic
	Presently exposed workers	26		20	<4-50	Spectrographic
	Workers not exposed for 10 to 20 years	35	8.6		3-22	Spectrographic
West Germany	Presently exposed workers	22	41			Atomic absorption after extraction

Source: Adapted from Friberg et al., Cadmium in the Environment, 2nd ed., Table 4:5, p. 56, (c) CRC Press, Inc., 1974. Used by permission of CRC Press, Inc.

and of occupationally exposed workers. Average cadmium levels in whole blood ranging up to 2 ppm have been reported (Butt et al., 1964); however, analytical errors are probable because even levels in exposed workers have been far lower (Table 6.5).

6.2.5.2 In Organs — As mentioned in Section 6.2.2.2, about 50% of the body burden of cadmium is found in liver and kidneys. A single "normal" concentration cannot be given for these organs because the values increase with age and vary among individuals, depending on diet, local environment, and smoking habits. For instance, the cadmium content of liver and kidneys as a function of age in various parts of the world is shown in Figures 6.4, 6.5, and 6.6. The decline in kidney cadmium levels after age 50 may reflect such factors as changing dietary and smoking habits, bias in the older groups with respect to disease and sex, and changes in cadmium content of foods. It could also result from overt renal damage as high tissue levels of cadmium are reached; the damaged organ might then leak cadmium into the urine.

Generally, the average body burden in a "standard man" (70 kg in weight) lies between 15 and 30 mg in the United States, 15 and 20 mg in the United Kingdom and Sweden, and 40 and 80 mg in Japan (Figures 6.4, 6.5, and 6.6). Median levels of cadmium in the pancreas have been reported as 1 ppm wet

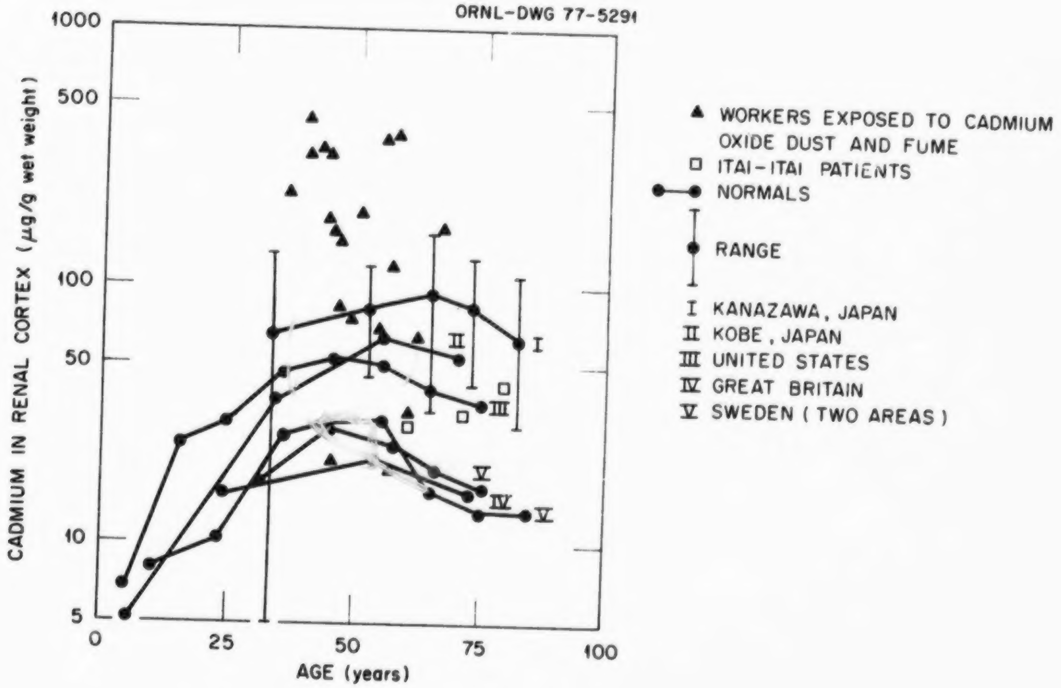


Figure 6.4. Cadmium content in renal cortex as a function of age, exposure, and geographical location. Source: Adapted from Friberg et al., Cadmium in the Environment, 2nd ed., Figure 4:22, p. 62, (c) CRC Press, Inc., 1974. Used by permission of CRC Press, Inc.

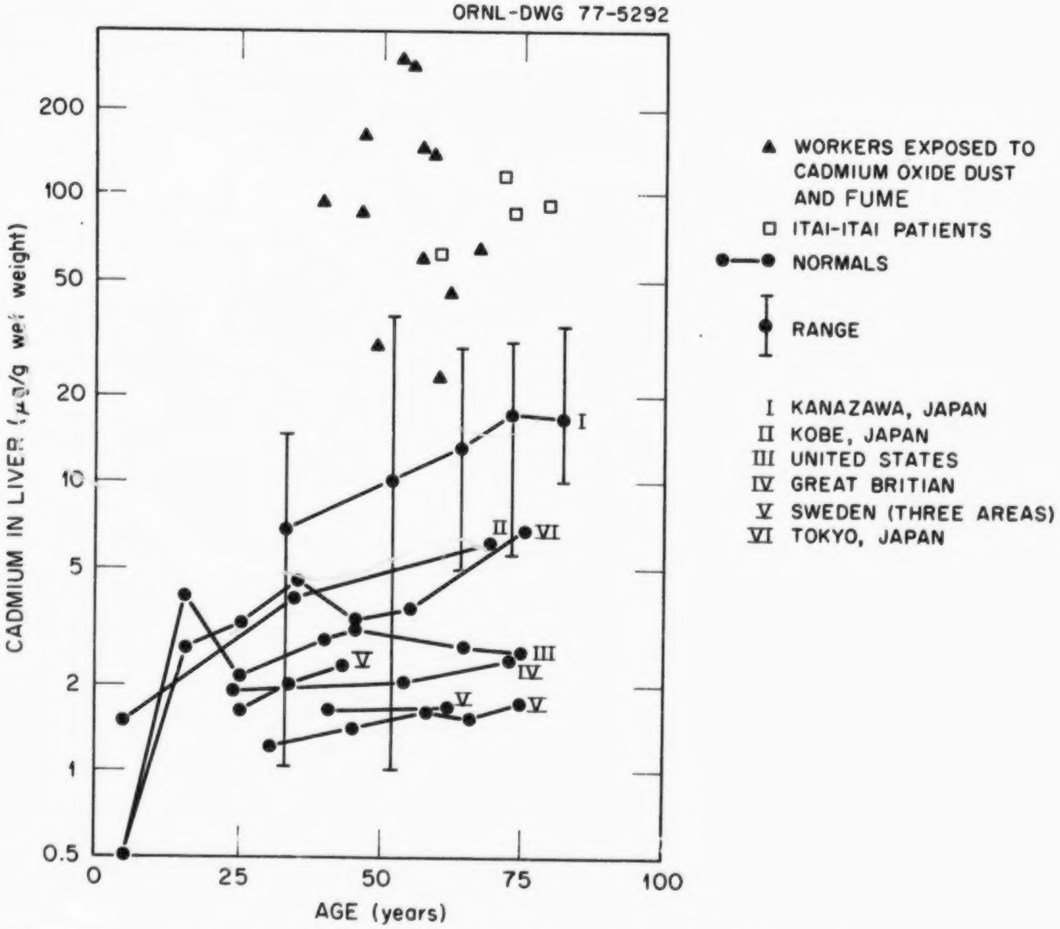


Figure 6.5. Cadmium content in liver as a function of age, exposure, and geographical location. Source: Adapted from Friberg et al., Cadmium in the Environment, 2nd ed., Figure 4:24, p. 63, (c) CRC Press, Inc., 1974. Used by permission of CRC Press, Inc.

weight in the United States (Tipton and Cook, 1963) and 2.8 ppm wet weight in the Far East (Tipton et al., 1965). A mean cadmium level in the lungs of San Francisco residents of 0.34 ppm wet weight was found by Tipton and Shafer (1964); this value compared with a mean level of 0.26 ppm wet weight found in 21 Scottish persons (40 to 70 years old) (Molokhia and Smith, 1967). Other lung values reported (Smith et al., 1960, as cited in Friberg et al., 1974) ranged up to 0.71 ppm wet weight in three British samples (mean age, 54 years). Lung concentrations of cadmium seem to increase with age. Small amounts of cadmium are found in other organs (Table 6.1).

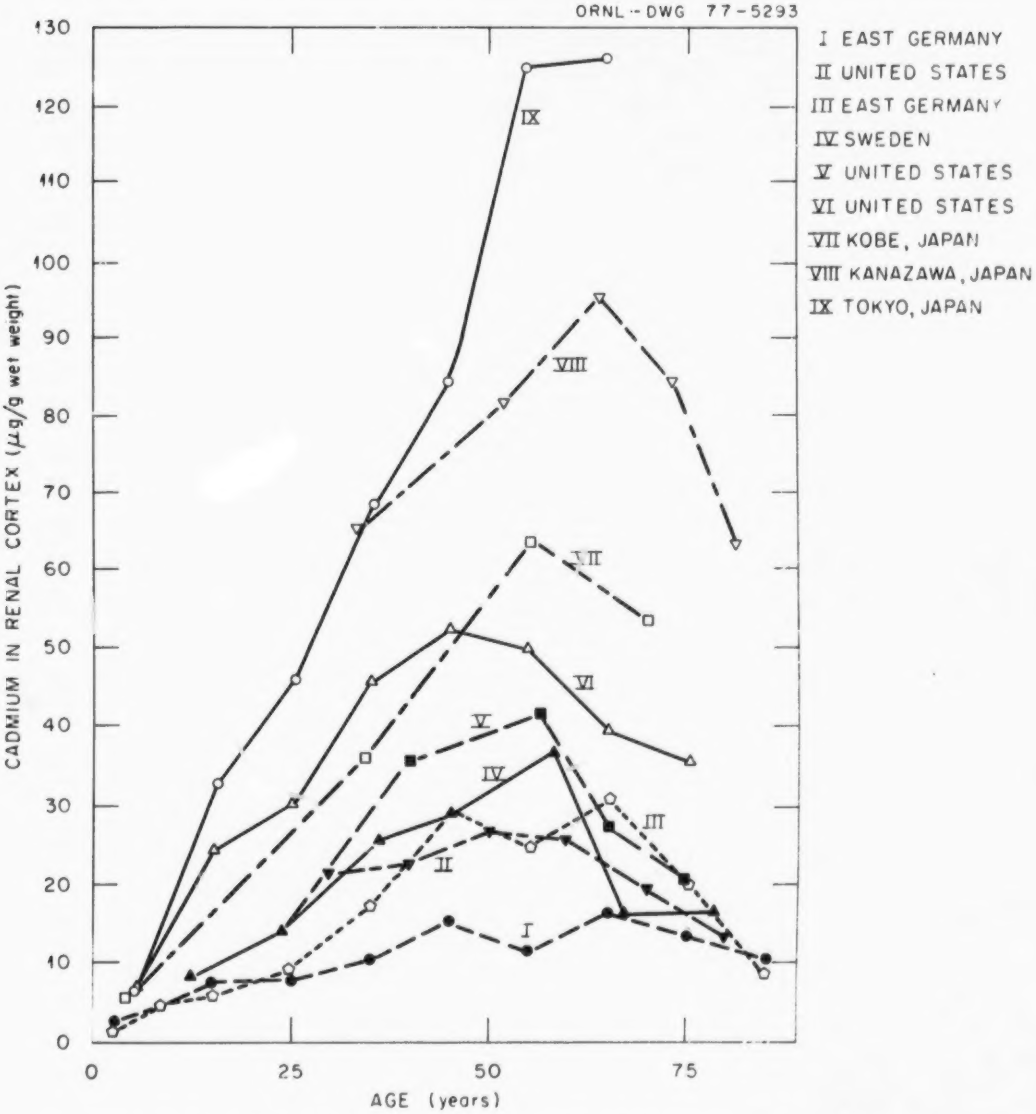


Figure 6.6. Cadmium content in renal cortex as a function of age and geographical location. Source: Adapted from Friberg et al., Cadmium in the Environment, 2nd ed., Figure 4:25, p. 64, (c) CRC Press, Inc., 1974. Used by permission of CRC Press, Inc.

6.2.6 Blood, Urine, and Feces as Indicators of Body Burden

For practical purposes, blood cadmium levels are not good indicators of organ cadmium levels, although blood levels may be higher in exposed workers, especially during exposure, than in unexposed persons. As an example, two Swedish factory workers (employed 20 years) not exposed directly to cadmium had blood cadmium levels of 0.021 and 0.031 ppm with renal damage, whereas two workers directly exposed to cadmium (three to four years of exposure) had blood cadmium levels of 0.048 and 0.058 ppm without renal damage (Piscator, 1971, as cited in Friberg et al., 1974). The equivocal nature of available data makes meaningful conclusions impossible.

During long-term, low-level exposure to cadmium, urinary cadmium levels are related to body burden on a group basis; however, the wide range of recorded values makes the relationship less meaningful for predicting organ levels on an individual basis (Nordberg, 1972). A relationship probably does not exist between urinary cadmium and body burden in occupationally exposed workers (Section 6.2.3.1). Available data do not permit conclusions on a relationship between fecal cadmium excretion and body burden.

6.3 EFFECTS

6.3.1 Mechanisms of Action

Before specific systemic effects produced by acute or chronic exposure to cadmium are discussed, some possible mechanisms of action at the cellular and molecular levels are mentioned.

6.3.1.1 Metallothionein — Metallothionein (the protein thionein plus a metal) seems to be intimately involved in cadmium metabolism (Section 2.3.4.2). This low-molecular-weight protein has the ability to bind cadmium, zinc, mercury, copper, silver, tin, or iron due to a high cysteine content and a resultant large number of sulfhydryl groups (Sabbioni and Marafante, 1975). A good discussion of metallothionein is presented in Friberg et al. (1974).

Actually, more than one species of metallothionein from different organs can be distinguished. Various functions have been assigned to the protein, including roles in cadmium transport in blood, in the organ distribution of cadmium, and in its excretion (Nordberg, 1972). These suggested functions, while plausible, have not all been critically tested. Quantitative analyses of the renal handling of hepatic cadmium metallothionein, for instance, have made it appear unlikely that this compound contributes to cadmium excretion in rabbits (Nomiyama and Foulkes, 1977).

A protective role for metallothionein has also been proposed. Injection of small doses of cadmium prior to a normally lethal dose of the metal prevents death, presumably by inducing synthesis of the metal-binding protein in liver and other organs (Nordberg, 1972; Piscator and Axelsson, 1970; Terhaar et al., 1965). Metallothionein is also less toxic to the testes than is an equivalent amount of unbound cadmium (Nordberg, 1971).

In addition, Suda et al. (1974) observed that, unlike the free metal, metallothionein-bound cadmium did not inhibit activation of vitamin D by kidney mitochondria.

On the other hand, metallothionein-bound cadmium is more toxic to the kidneys than are cadmium salts injected as such (Nordberg, Goyer, and Nordberg, 1975). High toxicity of cadmium metallothionein was also described by Webb (1975). The high nephrotoxicity of metallothionein as well as of other low-molecular-weight complexes of cadmium may be explained by the fact that such complexes are readily taken up by the kidney (Cherian, Goyer, and Delaquerriere-Richardson, 1976; Foulkes, 1974; Nordberg, Goyer, and Nordberg, 1975). Inorganic cadmium, in contrast, is neither filtered nor accumulated by the kidney (Foulkes, 1974). The selective accumulation of cadmium in the renal cortex may be related to the uptake of circulating metallothionein (Piscator, 1964, cited in Friberg et al., 1974).

Syversen (1975) isolated a cadmium-binding protein from liver and kidneys in 19 of 40 human autopsy samples. The mean cadmium level in the kidneys of the 19 was significantly higher than the mean of all 40 together. Table 6.6 gives cadmium and zinc levels in kidney and liver as a function of metallothionein content in these samples.

6.3.1.2 Enzyme Effects — Just as cadmium can combine with the sulfhydryl groups in metallothionein (thionein), it also reacts with the sulfhydryl groups in some mitochondrial and extramitochondrial enzymes. In mitochondria, according to Berry, Osgood, and St. John (1974), the metal is active in at least three places: (1) it binds to sulfhydryl groups of enzymes necessary for transfer of electrons from compounds in the citric acid cycle to compounds in the electron transport chain; (2) cadmium ion binds to and inactivates one or more enzymes necessary for the synthesis of adenosine triphosphate (ATP); and (3) cadmium ion also binds to the enzyme adenosine triphosphatase (ATPase), which is required to convert ATP to ADP (adenosine diphosphate) + PO_4, an important source of energy in cellular reactions.

Jacobs et al. (1956) showed that cadmium at concentrations as low as 5×10^{-6} *M* completely inhibits the phosphorylation coupled to oxidation of succinate or citrate in rat liver mitochondria. Similarly, Mustafa, Cross, and Tyler (1971) and Mustafa et al. (1971) showed that cadmium depresses respiration of alveolar macrophages. Cadmium completely inhibits uptake of oxygen by macrophage mitochondria at 5×10^{-5} *M* and uncouples oxidative phosphorylation at 5×10^{-6} *M*. Adenosine triphosphatase activity is also inhibited. Interference with respiration probably results from Cd^{2+} binding to flavoproteins and other dehydrogenases. Cadmium ions bind to cell membranes and may cause alterations in membrane integrity with resulting effects on cellular metabolism (Mustafa, Cross, and Tyler, 1971). Cadmium injected intraperitoneally into rats was subsequently found in purified liver ribosomes and enzyme preparations, indicating the presence of bound metal (Norton and Kench, 1977). Incorporation of amino acids into proteins was depressed, providing a possible explanation for the occurrence of lower-molecular-weight peptide chains and proteins in cadmium intoxication (Section 6.3.1.1).

TABLE 6.6. CADMIUM AND ZINC VALUES IN HUMAN LIVER AND KIDNEY

Sample	Number of samples	Age (years)		Smokers (%)	Died from cardio-vascular disease (%)	Liver content (ppm, dry wt)			Kidney content (ppm, dry wt)					
									Cortex			Medulla		
		Mean	Range			Cd	Zn	Cd/Zn	Cd	Zn	Cd/Zn	Cd	Zn	Cd/Zn
Reference samples from patients in which no metallothionein was detected	21	70	50–82	47	55	6.3 ± 4.9	298 ± 183	0.021	83 ± .5	237 ± 85	0.350	49 ± 19	165 ± 55	0.297
Autopsy samples with small amount of metallothionein	14	59	43–91	64	57	9.1 ± 4.0	298 ± 126	0.032	152 ± 20 (P < 0.0005)	280 ± 58	0.543	95 ± 28 (P < 0.0005)	221 ± 69	0.430
Autopsy samples with relatively large amount of metallothionein	5	60	54–74	80	80	12.2 ± 4.2	346 ± 108 (P < 0.025)	0.035	219 ± 51 (P < 0.0005)	206 ± 40	0.740	147 ± 21 (P < 0.0005)	275 ± 64	0.535

Source: Adapted from Syverson, Arch. Environ. Health, March 1975, Vol. 30, p. 160. Copyright 1975, American Medical Association. Reprinted by permission of the publisher.

Some cadmium effects may be due to the substitution of cadmium for zinc in zinc-requiring enzymes (Smith, 1973). Griffin et al. (1973) found that replacement of zinc by cadmium in aspartate transcarbamylase (from *Escherichia coli*) altered the conformation of the metal-binding site, which in turn altered the conformation of the active site.

Because cadmium can produce some toxic effects by combining with sulfhydryl groups in enzymes and by replacing zinc in zinc-requiring enzymes, it is not surprising that some acute effects of cadmium poisoning can be prevented or reduced by addition of zinc or sulfhydryl compounds, which act as competitive inhibitors of the cadmium-enzyme reactions (Gunn, Gould, and Anderson, 1968; Parizek, 1957; Parizek and Zahor, 1956). Selenium is also an effective cadmium antagonist (Gunn, Gould, and Anderson, 1968). Chen, Whanger, and Weswig (1975) suggested that selenium counteracts the toxicity of metals by altering their tissue concentration and by diverting them to presumably less critical components. Parizek (1957) found that the testicular damage induced in rats by a subcutaneous injection of 0.04 millimole cadmium chloride per kilogram body weight could be completely prevented by injecting 80 to 200 times that amount of zinc acetate divided into three doses — one given 5 hr before cadmium, one with the cadmium, and one 19 hr after cadmium. Other effects of cadmium, such as anemia and weight loss, have been corrected in rats and mice by administration of zinc (Bunn and Matrone, 1966; Hill et al., 1963). Similar results were obtained in other studies (Friberg et al., 1974). Rats given a diet with sufficient zinc suffered typical symptoms of zinc deficiency if an equimolar amount of cadmium was also given. The zinc-to-cadmium ratio may be as important as the absolute amount of either (Voors, Shuman, and Gallagher, 1973). The work of Webb (1972) suggested that the protective effect of preliminary injections of zinc can be accounted for by the zinc-induced synthesis of a protein with a high binding affinity for cadmium (metallothionein).

6.3.2 Toxic Effects

Several good reviews of cadmium toxicity are available (Flick, Kraybill, and Dimitroff, 1971; Friberg et al., 1974; Fulkerson and Goeller, 1973). Acute or chronic cadmium poisoning can occur through ingestion, inhalation, or both. The major problem for human populations is chronic, low-level exposure through ingestion, although occasional acute poisoning via this route has occurred. Workers in cadmium-related industries may be chronically exposed to higher cadmium levels in the air than the general population and also have a greater chance of acute poisoning through accidents.

6.3.2.1 Effects in Livestock — Miller et al. (1967) gave 3 g of cadmium daily for two weeks to holstein cows but produced only minimal effects. Consumption of a food concentrate dropped on the second day of cadmium administration, remained at less than one-half the pre-cadmium level on the third and fourth days, and then increased to normal by the eighth day even though cadmium was still being administered. Effect on total food intake was not measured; however, the reduced concentrate consumption reflected a systemic effect on appetite rather than a change in palatability of the food because cadmium was not mixed with the food. The cows lost an average of 30 kg during cadmium treatment and regained 5 kg during three weeks after cadmium treatment stopped. The depression of milk synthesis was more than a simple result of decreased concentrate consumption; two weeks after cadmium was stopped, milk production was still only 73% of that before cadmium, as shown in Figure 6.7. The controls had decreased to 82% of initial milk production during this time; the small number of experimental animals (three) makes the significance of the difference questionable. At the time of greatest depression of milk synthesis, fat content of the milk increased about 44% and then returned to normal after cadmium administration stopped. No other cadmium-related effects were found. Other discussions of cadmium excretion into milk are found in Dorn et al. (1973), Lynch, Cornell, and Smith (1974), McClanahan et al. (1965), and in Section 6.2.3.4.

ORNL-DWG 77-5287

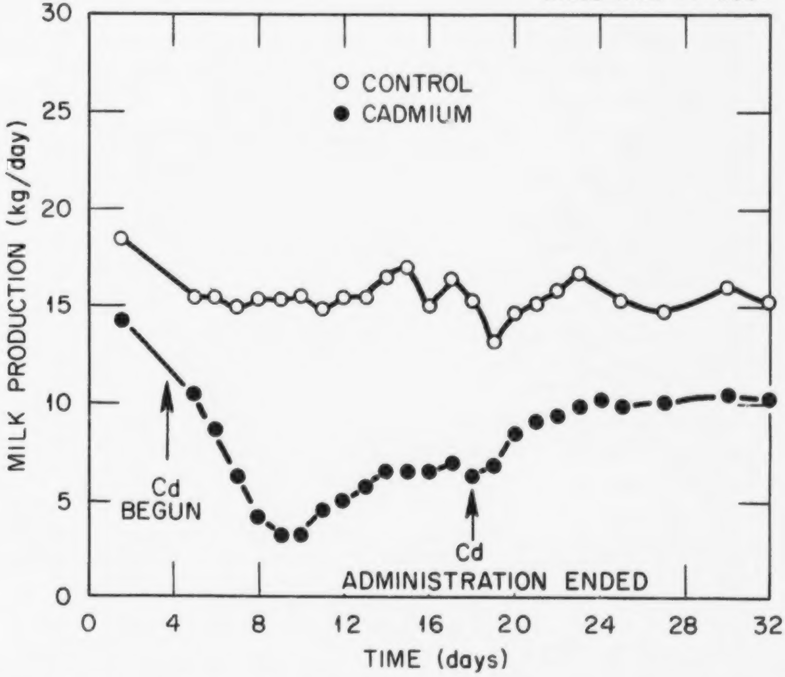


Figure 6.7. Effect of oral cadmium administration on milk production of lactating holstein cows. Source: Adapted from Miller et al., 1967, Figure 2, p. 1406. Reprinted by permission of the publisher.

Cadmium poisoning of domestic livestock should rarely occur because most sources of possible contamination are recognized and can be controlled. Unfortunately, this has not been achieved in fact. According to Aschbacher (1973), there were no reported toxicities in livestock from cadmium, but Goodman and Roberts (1971) reported sporadic livestock illness downwind of Swansea (southeast Wales), an area contaminated by copper, zinc, lead, and cadmium. A horse which had eaten hay grown in the area developed lead poisoning; when it died, tissue analysis revealed a renal cadmium level of 330 ppm wet weight, a level almost certainly associated with renal damage, regardless of the immediate cause of death.

Tissues from nine cattle living about 15 miles from a zinc smelter in Montana were examined (Munshower, 1972). Cadmium levels in the kidneys and liver averaged 1.67 and 0.34 ppm (wet weight) respectively. The cadmium concentration in these organs appeared to increase with age. A similar situation existed around a smelting operation in East Helena, Montana (U.S. Environmental Protection Agency, 1972). The hair of mammals in this area had significantly higher cadmium concentrations than hair from animals in other areas. Livestock in the area, especially horses, showed signs of heavy-metal poisoning and had high cadmium and lead concentrations in their kidneys and livers. Other reports of livestock poisoning are cited by Smith and Huckabee (1973). Although exposure usually included several metals which could interact, cadmium levels were high in all cases.

6.3.2.2 Acute Cadmium Poisoning

6.3.2.2.1 Inhalation — Acute cadmium poisoning through inhalation primarily affects industrial workers (e.g., welders and metal workers) because cadmium is present in some silver solders, brazers, and platings on various metals. The high temperatures used in welding and other processes can cause formation of cadmium oxide fumes (Section 2.3.1). Many case histories of workers poisoned while welding or cutting cadmium-plated metals are reported in the literature (Friberg et al., 1974; Hise and Fulkerson, 1973). Usually, no discomfort occurs during acute exposure to cadmium fumes, and symptoms do not generally appear for 4 to 8 hr; a lethal amount of cadmium can therefore be absorbed without warning (Beton et al., 1966; Blejer, Caplan, and Alcocer, 1966). Symptoms of acute inhalation of cadmium include: (1) metallic taste in the mouth; (2) headache; (3) shortness of breath, chest pain, cough with foamy or bloody sputum, abnormal pulmonary rales, and physical signs which mimic the flu; (4) weakness and leg pains; (5) pulmonary edema, which may lead to death or may gradually improve over several days; (6) pneumonic consolidation; and (7) late liver damage (Hise and Fulkerson, 1973).

Freshly generated cadmium fume is the most toxic form of cadmium when inhaled (Princi, 1947). Fume generated from arc welding is twice as toxic as that thermally generated by oxyacetylene, possibly because of smaller particle sizes in arc-generated fume (Princi, 1947). The lethal time-dose of thermally generated fume (air concentration in milligrams multiplied by the exposure time in minutes) approaches 2900 min-mg/m^3 for humans, whereas the lethal time-dose of arc-generated fume probably does not exceed 1400 min-mg/m^3 (Barrett and Card, 1947). Lethal time-doses of thermally generated fume of 2500 and 2600 min-mg/m^3 have also been reported, corresponding to an 8-hr exposure to about 5 mg/m^3 (Beton et al., 1966). A cadmium concentration lower than 5 mg/m^3 for 8 hr might also be fatal since concentrations as low as one-fourth the LD$_{50}$ produced acute symptoms and permanent lung damage in animal experiments (Paterson, 1947).

Data are not available to permit calculation of lethal doses for cadmium dusts. In rabbits the lethal concentration of cadmium-iron dust is three to four times higher than that of cadmium oxide fumes (Friberg, 1950).

6.3.2.2.2 Ingestion — Acute cadmium poisoning due to ingestion by humans is rare since cadmium use near food or drinks (e.g., cadmium-plated food containers) is prohibited. Organic acids present in many foods and drinks dissolve cadmium, forming organic cadmium salts which dissolve in gastric juices (Hise and Fulkerson, 1973). Symptoms following cadmium ingestion are similar to those of food poisoning (Frant and Kleeman, 1941). Symptoms, which may begin one-half to one hour after ingestion, include: (1) severe nausea, vomiting, diarrhea, abdominal cramps, and salivation; (2) headache, muscular cramps, vertigo, and (rarely) convulsions; (3) exhaustion, collapse, shock, and death, usually within 24 hr, or gradual appearance of liver and kidney damage; and (4) death in one to two weeks from acute renal failure (Hise and Fulkerson, 1973).

Fortunately, cadmium is emetic when ingested, eliminating some cadmium before absorption can occur. The emetic threshold is probably in the range of 3 to 15 mg (Hise and Fulkerson, 1973). The same dose of cadmium may be more toxic in water than in food since some cadmium may bind to components in the food and be unavailable for absorption (U.S. Department of Health, Education, and Welfare, 1962). Differences in the amount of ingested cadmium bound to food may be responsible for some of the variability in reported toxic values. The following ranges of acute toxic values in humans have been reported (Hise and Fulkerson, 1973):

3 to 90 mg	Emetic threshold
11 mg	Reported nonfatal incidents
10 to 326 mg	Severe toxic symptoms (nonfatal)
350 to 3500 mg	Estimated lethal dose
8900 mg	Reported lethal dose

6.3.2.3 Chronic Cadmium Poisoning — Chronic cadmium poisoning is probably more important to the general population — and even to workers in cadmium-related industries — than acute poisoning because safeguards and restrictions on cadmium use make it unlikely that cadmium levels high enough to cause acute effects will be present. Normal background levels of cadmium in air, water, and food together with regionally higher levels from local industries and low-level occupational exposure make chronic poisoning more likely.

6.3.2.3.1 Inhalation — Chronic cadmium poisoning in industry is essentially an inhalation problem. The most prominent symptoms are chronic bronchitis, emphysema, and proteinuria (Friberg et al., 1974; Hise and Fulkerson, 1973; Nordberg, 1974). These effects have been produced by cadmium oxide fumes, cadmium oxide dust, and cadmium pigment dust; in general, they do not appear until after a few years of exposure (Friberg et al., 1974). Dose-response curves are difficult, if not impossible, to determine accurately because varying exposure conditions permit only estimates of actual doses.

Qualitative evidence of lung damage was obtained 25 years ago from workers in Sweden (Friberg, 1950). With an average employment time of 20 years, 43 male workers exposed to cadmium oxide dust had impaired lung function; a group of 15 workers with similar exposure but only one to four years of employment had normal lung function. Cadmium concentrations in air were reported as 3 to 15 mg/m^3, but measurements were only made once at five areas of the factory. Similar results were found at a German factory

(Baader, 1952). Lauwreys et al. (1974) observed renal damage in men exposed for over 20 years to a concentration of respirable cadmium of only 21 μg/m^3 (Section 6.3.2.3.1).

A group of 100 men exposed to cadmium oxide fumes and a group of 104 controls were examined by Bonnell (1955). Eleven of the exposed workers had emphysema, but no emphysema was mentioned for the controls. There were significant differences between the two groups, on a group basis, in part of the respiratory function test. The mean flow rates at 30, 50, and 70 respirations per minute were significantly lower in the exposed group, but the vital capacities and maximum ventilatory capacities were similar. No information on cadmium doses was given. Other studies of occupationally exposed workers show similar results, with equally poor dose information (Friberg et al., 1974). It is likely, however, that prolonged exposure to cadmium oxide fumes at levels below 0.1 mg/m^3 could lead to impairment of lung function.

Suzuki, Suzuki, and Ashizawa (1965) reported a study of workers exposed to cadmium stearate dust and lead. No differences in lung function tests between exposed workers and controls were observed. Time of employment was 3.3 years with a standard deviation of 1.9 years. Cadmium concentrations varied from 0.03 to 0.69 mg/m^3 at different operations; particle size varied from 0.4 to 20 μm. Exposure was for 20 min three or four times a day. The negative findings may reflect the short employment time.

6.3.2.3.2 Ingestion — The following toxic effects are discussed in this section because the general population is more likely to be affected by chronic ingestion of cadmium than by inhalation; however, it should be understood that chronic cadmium inhalation can also produce these effects. In addition, ingestion of cadmium also appears capable of producing pulmonary lesions (Vinegar and Choudhury, 1976).

6.3.2.3.2.1 Proteinuria — The kidney is the critical organ in cases of prolonged, low-level exposure to cadmium (Friberg et al., 1974; Nordberg, 1974). As mentioned previously (Sections 6.2.2.2 and 6.2.5.2), about one-third of the body burden of cadmium is found in the kidneys. When the cadmium concentration in the renal cortex reaches about 200 ppm wet weight, morphological changes and/or proteinuria may occur (Friberg et al., 1974; Nordberg, 1974). The usefulness of this value, which was determined from autopsy or biopsy samples of renal tissue from cadmium workers and which agrees with values determined in animal experiments, has been questioned by Nomiyama et al. (1977). Following onset of proteinuria, the cadmium level in the kidneys decreases as cadmium is excreted along with the protein (Friberg et al., 1974). Table 6.7 shows cadmium content in the renal cortex and morphological change in workers exposed to cadmium oxide dust. The lower cadmium values associated with the greatest morphological change probably reflect release of cadmium as mentioned above.

Proteinuria is a common finding in cadmium workers (Bonnell, 1955; Friberg, 1950; Kazantzis et al., 1963; Smith and Kench, 1957). Other renal changes, including glucosuria, amino aciduria, decreased concentrating capacity, and formation of renal stones, are common (Adams, Harrison, and

TABLE 6.7. CONCENTRATIONS OF CADMIUM IN KIDNEY CORTEX OF WORKERS EXPOSED TO CADMIUM OXIDE DUST IN RELATION TO MORPHOLOGICAL KIDNEY CHANGES SEEN AT AUTOPSY OR BIOPSY

Worker	Age	Years exposed	Years since last exposure	Year of autopsy or biopsy	Morphological changes	Proteinuria[a]	Cadmium in cortex (ppm wet wt)
S.W.H.	46	28	1	Autopsy, 1960	Profound	+	83[b]
K.J.	49	22	9	Autopsy, 1951	Profound	+	75[b]
K.N.	57	18	6	Autopsy, 1952	[c]	+	20[b]
H.B.	60	26	3	Autopsy, 1949	Profound	+	33[b]
A.B.	39	16	4	Autopsy, 1950	Profound	+	174[b]
O.J.	62	20	1	Autopsy, 1967	Profound	+	63
G.J.	44	11	12	Biopsy, 1959	Slight	+	321
G.K.	46	15	0	Biopsy, 1959	Slight	+	152
A.L.	36	6	0	Biopsy, 1959	None	(+)	220
E.Y.	39	7	0	Biopsy, 1959	None	-	446
E.H.	40	15	10	Biopsy, 1959	None	+	320
J.P.	43	20	6	Biopsy, 1959	None	+	330
N.U.	44	12	0	Biopsy, 1959	None	-	180
H.N.	45	13	0	Biopsy, 1959	None	+	21
K.N.	50	15	2	Biopsy, 1959	None	-	190

[a] - — negative results on repeated testing with trichloroacetic acid.
(+) — varying results.
+ — positive results on repeated testing.

[b] Figures based on cadmium concentration in whole kidney, assuming that the cadmium concentration in cortex is 1.5 times the average kidney concentration.

[c] Results from histological examinations not reported, but in an examination in 1946 this worker had the lowest kidney function tests of all (inulin clearance, 42 ml/min; maximum specific gravity of urine, 1016, blood nonprotein in nitrogen, 44 mg %).

Source: Adapted from Friberg et al., Cadmium in the Environment, 2nd ed., Table 6:1, p. 108, (c) CRC Press, Inc., 1974. Used by permission of CRC Press, Inc.

Scott, 1969; Ahlmark et al., 1961; Friberg, 1950; Kazantzis et al., 1963; Piscator, 1966). Forty-four percent of a group of workers exposed to cadmium dust for more than 15 years had a history of renal stones (Ahlmark et al., 1961).

Lauwerys et al. (1974) surveyed three groups of workers exposed to cadmium dusts (Group I: women with less than 20 years of exposure; Group II: men with less than 20 years; and Group III: men exposed for longer than 20 years). Group III was exposed to a respirable cadmium concentration of only 21 μg/m³; these workers showed slight pulmonary but more pronounced renal symptoms (proteinuria). The theoretical calculations of Friberg et al. (1974) suggest that even lower concentrations of cadmium in ambient air could lead over a period of 50 years to critical cadmium levels in the renal cortex.

6.3.2.3.2.2 Anemia — Although kidney damage is usually the limiting factor in chronic cadmium exposure, the hematopoietic system is also affected

(Friberg et al., 1974; Hise and Fulkerson, 1973; Nordberg, 1974). Moderate anemia has been found in workers exposed to cadmium oxide dust and fumes (Friberg, 1950; Tsuchiya, 1967). A significant correlation between high cadmium levels in blood and low or below normal hemoglobin levels was found in 16 workers with 5 to 30 years exposure; the mean haptoglobin level was below normal, indicating hemolysis (Piscator, 1971, as cited in Friberg et al., 1974). The anemia is apparently reversible since workers with no exposure for at least ten years had normal or elevated haptoglobin levels (Piscator, 1971, as cited in Friberg et al., 1974). As in many other epidemiological studies, dose-response relationships are impossible to calculate.

Cadmium-induced anemia has been produced experimentally in animals including rats, rabbits, and Japanese quail (Decker et al., 1958; Fox and Fry, 1970; Friberg, 1950; Piscator and Axelsson, 1970). In rats, 31 ppm cadmium in food for several months produced anemia; 125 ppm cadmium for seven months also produced an increase in reticulocytes and eosinophils and induced hyperplasia of the bone marrow (Wilson, DeEds, and Cox, 1941). After exposure ended, hemoglobin levels returned towards normal.

Decker et al. (1958) found no pathological changes in the blood of Sprague-Dawley rats given up to 10.0 ppm cadmium in water for one year; however, at a level of 50.0 ppm cadmium in water a significant decrease in hemoglobin was apparent in two weeks. Hemoglobin dropped to 8 g/100 ml of blood from an average of 15.5 g/100 ml in controls and remained there for the duration of the study. Anemia may be partly an iron-deficiency anemia due to decreased uptake of iron from the intestines, partly a result of increased plasma volume, and partly a hemolytic anemia (Axelsson and Piscator, 1966; Berlin and Piscator, 1961; Fox and Fry, 1970). Iron supplementation prevented anemia as did ascorbic acid, which probably acted by increasing iron absorption from the intestines (Fox and Fry, 1970; Pond and Walker, 1972; Sansi and Pond, 1974). Pond, Walker, and Kirtland (1973) reported that anemia induced in weanling Yorkshire pigs by 154 ppm cadmium (as $CdCl_2$) in the diet could be prevented by oral administration of 400 ppm iron (as $FeSO_4 \cdot 7H_2O$) throughout the experiment or 1000 mg iron (as iron dextran) in a single intramuscular injection on the first day of the experiment. Chatterjee, Banerjee, and Rudra Pal (1973) showed that anemia induced in rats by 60 mg/kg body weight daily of $CdCl_2 \cdot H_2O$ could be prevented by the addition of 1.0 mg/kg body weight daily of L-ascorbic acid along with the cadmium.

6.3.2.3.2.3 Hypertension — The question whether cadmium can cause hypertension in humans remains unanswered. Hypertension can be induced in some animals by cadmium treatment, and epidemiological studies have provided some support for the existence of cadmium-induced hypertension (Friberg et al., 1974). The evidence is not conclusive, however. Cadmium-induced hypertension is reviewed also in Hise and Fulkerson (1973).

In general, some epidemiological data show a correlation between cadmium and hypertension in humans; however, these data are ambiguous and certainly are not proof of a causal relationship. For example, Carroll (1966) reported a positive correlation between cadmium in air and death rates from hypertension and arteriosclerotic heart disease in 28 American cities (coefficient of correlation = 0.76). Several years later Carroll's data were reanalyzed by other researchers who found an even higher correlation between population density and hypertension than between cadmium and hypertension (Hunt et al., 1971). In an area with an average urban cadmium level in ambient air (0.01 μg/m^3), the estimated uptake over a 50-year period would be only 1 mg, a small fraction of the total body burden (Sections 6.2.1.1 and 6.2.5).

Schroeder (1965) reported a correlation between cadmium in the kidneys and the relative incidence of hypertension in about 400 persons from cities around the world. Similar studies are reported in Friberg et al. (1974) and Hise and Fulkerson (1973); however, factors such as population density, smoking, and disease history, which may also be related to hypertension, are not discussed. Further, several studies failed to find a relationship between kidney cadmium levels and hypertension (Hunt et al., 1971; Lewis et al., 1972*a*; Morgan, 1969). In addition, there is no evidence that workers in cadmium-related industries have a higher incidence of hypertension than does the general population. Patients in Japan with itai-itai disease and persons living near the endemic area do not have an unusually high incidence of hypertension (Friberg et al., 1974).

An inverse correlation between hypertension and the hardness of drinking water has been reported (Crawford, Gardner, and Morris, 1968; Morris, Crawford, and Heady, 1961). Hard water usually contains less cadmium than soft water (Section 2.3.2.1.1). Soft or acid waters may contain cadmium levels above the U.S. Public Health Service limits (0.01 ppm) (Schroeder et al., 1967) (Table 6.8). In a survey of 969 public water supply systems, less than 1% exceeded 0.01 ppm (McCabe et al., 1970). Most studies found this inverse correlation between hypertension and water hardness; however, one study by Morton (1971) and a controversial study by Bierenbaum (1975) and Bierenbaum et al. (1975) showed a positive correlation. In the latter study, Kansas City, Kansas, was reported to have twice the cardiovascular death rate and a higher average blood pressure than Kansas City, Missouri. Both cities use the same source of water, but Kansas City, Missouri, softens its water to about one-half. While this result seems to contradict most other studies, the hard water contained three times as much cadmium as the soft water, and the serum from hard-water drinkers contained 13 times as much cadmium as that from soft-water drinkers (16.4 ppb versus 1.2 ppb). The attribution of a 13-fold higher cadmium concentration in serum to a three-fold increase in water cadmium, as presented in this study, is highly controversial. In fact, the validity of the measurements in the study has been questioned (Sharrett and Feinleib, 1975). Also, the possibility of other sources of cadmium in the populations investigated were not considered in this study. In addition, zinc is known to reverse cadmium-induced hypertension in animals (Schroeder and Buckman, 1967) and serum zinc levels in the area of the Kansas City study with lower cadmium and lower blood pressure were three times higher than in the area with high cadmium levels and elevated blood pressure. The cadmium-zinc ratio may be more important than the content of cadmium alone (Schroeder et al., 1967). Cadmium could conceivably affect the kidney at doses too low to produce obvious toxicity and the resulting increase in sodium retention might raise the blood pressure. The Kansas City study appears to be consistent with, but does not prove, the hypothesis that cadmium can induce hypertension in humans.

TABLE 6.8. ZINC AND CADMIUM CONCENTRATIONS IN WATER AND SNOW

Sample type	Zinc (ppb)	Cadmium (ppb)
Natural water		
Connecticut River, Vt.	5	14.6[a]
Brook, Vt.	14	0.5
	3.5	0.6
Spring, N.H.	177	2.5
Seawater, Caribbean	24	0.3
Municipal water, Brattleboro, Vt., reservoir		
Inlet	3.5	2.1
Spillway	3.5	2.5
Main, town		14.0-21.0[a]
Tap, hospital		
Cold, running	160	8.3
Stagnant	1830	15.0-77.0[a]
Hot, running		21.0[a]
Institution, cold, running		1.0
Bridgeport, Conn., tap		14.0[a]
White Plains, N.Y., tap		14.0[a]
Bangor (Roseto), Pa.		0.0
Private dwellings		
Lake, N.H.	3.5	1.1
Tap, plastic pipe	20	14.0[a]
Spring, Vt.		3.8
Tap, iron pipe		3.5
Spring, Vt., copper pipe	770	8.3
Spring, Vt.	13	2.2
Tap, iron pipe	660	3.8
Well, Vt., iron pipe	100	1.1
	2160	3.5
Well, Vt., artesian, copper and plastic pipe	219	12.0[a]
Spring, S.C., iron pipe	18	8.0
Spring, Ark., bottled	<0.3	<0.3
Miscellaneous		
Distilled, glass still	6	0.5
Deionized	0	0
Snow, melted, 20 m from street, town, moderate traffic	1380	1.5
100 m from road, forest, hilltop	45	0.35
50 m from road, hilltop, behind laboratory	8	0.370

[a] Cause for rejection of water supply exceeding allowable limits of 10 ppb. Two copper pipes, dissolved in acid, contained 57 and 2.76 ppm cadmium. Galvanized iron pipes contained 140 to 400 ppm cadmium (mean = 300 ppm).

Source: Adapted from Schroeder et al., 1967, Table 10, p. 198. Reprinted by permission of the publisher.

Probably the main reason why a correlation has been sought between cadmium and hypertension in humans is that cadmium has been reported to induce hypertension in some experimental animals (Friberg et al., 1974; Hise and Fulkerson, 1973). Schroeder (1964) induced hypertension in Long-Evans rats with 5 ppm cadmium in their drinking water. About 17% of the males were hypertensive at 17 months; the percentage increased with age until 65% were hypertensive at 30 months. The incidence of hypertension was more constant in females, varying from 67% to 83%. The results of this study were possibly affected by intake of NaCl, although Schroeder concluded that the salt intake had little or no effect on hypertension in this study. Kidney and liver cadmium levels averaged 40 and 6 ppm wet weight, respectively, in another study by Schroeder, Nason, and Balassa (1967). These levels are comparable to those in adult North Americans. Animals with a cadmium-to-zinc weight ratio in the kidney of 0.8 or above were always hypertensive (Schroeder and Buckman, 1967).

Hypertension has also been produced in rats and rabbits by parenteral or intraperitoneal injections of cadmium (Schroeder and Buckman, 1967; Schroeder, Nason, and Mitchener, 1968; Thind et al., 1970). For example, 1 or 2 mg cadmium per kilogram body weight injected intraperitoneally as cadmium citrate into rats produced hypertension within a month. Blood pressure of hypertensive rats returned to normal within 300 days following cessation of cadmium exposure (Schroeder, Nason, and Mitchener, 1968). In some experiments, hypertension was not induced in rats by ingested (Lener and Bibr, 1970) or injected cadmium (Castenfors and Piscator, unpublished, as cited in Friberg et al., 1974). According to Friberg et al. (1974), strain differences and sodium intake may influence development of hypertension in experimental animals. Porter, Miya, and Bousquet (1974) were unable to produce hypertension in rats at various dose levels of cadmium and under various treatment schedules and conditions. Some subtle dietary or other environmental variables may be of major significance in the production of experimental hypertension with cadmium.

It is interesting to note that intraperitoneally injected cadmium in rats, and to a lesser extent mercury, has been found to increase both apparent renin activity in peripheral blood and blood pressure (Perry and Erlanger, 1973). For intraperitoneally injected cadmium, the increase in renin activity following a single dose of about 200 μg Cd^{+2} was evident within 1 min and persisted for one month (Perry and Erlanger, 1973). Perry and Erlanger (1973) also noted that in the case of "fed" cadmium, continuous exposure to 5 ppm in drinking water increased the renin activity of rats within a week; the level remained elevated after one month, but not after three months.

In summary, although cadmium can induce hypertension in animals and some correlation has been found between cadmium and hypertension in humans, no clear evidence directly implicates cadmium in human hypertension.

6.3.2.4 Other Effects

6.3.2.4.1 Liver function — An effect of cadmium on liver function has been seen in several workers with acute cadmium poisoning from cadmium oxide fumes; however, liver disturbances are not a common finding (Friberg et al., 1974). As mentioned in Section 6.2.2.2, cadmium concentration in the liver can be quite high during acute exposure, even higher than in the kidney. The glutamic oxaloacetic transaminase activity in rabbit serum was increased following intravenous injection of cadmium (Andreuzzi and Odesalchi, 1958, cited in Friberg et al., 1974) (Table 6.9). The authors concluded that a dose near the LD_{50} was necessary to produce severe liver lesions; at lower doses hepatic changes were reversible.

TABLE 6.9. CHANGES IN GLUTAMIC OXALOACETIC TRANSAMINASE ACTIVITY FOLLOWING INTRAVENOUS INJECTION OF CADMIUM INTO RABBITS

Dose (mg/kg)	Change in glutamic oxaloacetic transaminase activity	Remarks
3	Considerable increase after 17 hr	60% of animals dead in 24 hr
2.5	Tenfold increase after 24 hr	40% of animals dead in 48 hr
2	Fourfold increase after 24 hr; back to normal after 72 hr	All survived over 72 hr.
1.25	Fourfold increase after 24 hr; back to normal after 72 hr	All survived over 72 hr.

Source: Compiled from Friberg et al., 1974, p. 122.

Gross changes in liver function are rarely found in chronically exposed workers; however, animal studies have shown that morphological liver changes can be present when various liver function tests such as activities of glutamic oxaloacetic transaminase, alkaline phosphatase, and lactic dehydrogenase are within normal limits. These tests therefore may be poor indicators of cadmium damage to the liver (Stowe, Wilson, and Goyer, 1972). Several animal experiments, including those of Axelsson and Piscator (1966), Singhal et al. (1974), and Sporn et al. (1970, cited in Friberg et al., 1974) show effects of cadmium on liver enzymes.

6.3.2.4.2 Testicular damage — Testicular damage is also seen in animals following exposure to cadmium. In general, testicular damage occurred in animals with scrotal testes, but not in animals with abdominal testes. Strain differences in sensitivity among mice were demonstrated by Gunn, Gould, and Anderson (1965). Among 19 strains tested, testicular necrosis was consistently produced in 11 by subcutaneous injection of 3.3 mg/kg cadmium chloride. Testes of seven strains were unaffected even when the dose was raised to the lethal level (value not reported). Strain differences with respect to dose required to produce minimal testicular damage were also found in rats. The necrosis is probably due to vascular damage. Data on testicular effects associated with chronic cadmium exposure are inconclusive; possibly, induction of metallothionein, which has been shown to protect animals against acute cadmium-induced testicular damage, prevents testicular damage during chronic exposure (Nordberg, 1971; Piscator and Axelsson, 1970). Testicular necrosis has also been prevented by zinc, selenium, cobalt, and thiol compounds (Gabbiani, Baic, and Déziel, 1967; Gunn, Gould, and Anderson, 1963, 1966, 1968; Parizek, 1957).

On the basis of animal data (Smith et al., 1960, as cited in Friberg et al., 1974), testicular necrosis might also be expected in humans, but it has not been clearly demonstrated following either acute or chronic cadmium exposure. Some unspecific histological changes in the testes were found at autopsy in workers exposed to cadmium fume. The authors felt that the changes resulted from the terminal illnesses rather than from cadmium exposure. Abnormally low testosterone levels in the urine and impotence were found in one of ten workers examined in a 1968 study (Favino et al., 1968).

6.3.2.4.3 Insulin production – Cadmium accumulates in the pancreas, although to a minor extent compared with the kidneys or liver. Tipton and Cook (1963) reported a median value of 80 ppm in pancreatic tissue ash (approximately 1.0 ppm wet weight) from adults living in the United States. Tipton et al. (1965) reported median values of less than 50 ppm of ash in African subjects, 91 ppm of ash in Near Eastern subjects, and 230 ppm of ash in Far Eastern subjects. Since cadmium is present in the pancreas, an effect on insulin production is a possibility, but direct evidence is lacking. Intravenous administration of cadmium produces hyperglycemia in rabbits (Voinar, 1952) and in dogs (Caujolle et al., 1964). Blood glucose concentrations rose in mice following a single intraperitoneal injection of cadmium (2.0 to 6.0 mg/kg), and concentrations of circulating insulin decreased (Ghafghazi and Mennear, 1975). Ithakissios et al. (1975) showed that chronic cadmium administration to rats inhibited pancreatic secretory activity. Also, decreased pancreatic function is associated with itai-itai disease in humans (Murata et al., 1970).

Ghafghazi and Mennear (1975) perfused isolated rat pancreata with either glucose alone (300 mg/100 ml) or glucose in combination with 10^{-4} to 10^{-3} M cadmium. Glucose alone produced a rapid release of insulin; the secretion was significantly depressed by the lowest concentration of cadmium used and was completely inhibited by the higher concentrations. Inhibition was not reversed by subsequent perfusion with cadmium-free glucose solution (60 mg/100 ml) but was partially reversed by perfusion with glucose plus theophylline. On the basis of the work of Brisson, Malaisse-Lagae, and Malaisse (1972) the theophylline effect was taken to indicate a possible involvement of calcium ions.

Ithakissios et al. (1975) also found an inhibition of insulin secretion in perfused pancreata removed from rats that had received 70 intraperitoneal doses of cadmium (0.50 mg/kg dose every other day). Doses of 0.25 mg/kg did not cause a significant inhibition. The cadmium level in the pancreas was about 54 ppm wet weight in the rat receiving the higher dose, a value comparable to that reported in human itai-itai patients (45 to 65 ppm) (Ishizaki, Fukushima, and Sakamoto, 1970). The significance of this value is unclear since the rats receiving the low dose and showing little or no inhibition of insulin secretion actually had higher cadmium levels (approximately 76 ppm wet weight).

6.3.2.4.4 Carcinogenesis -- No direct evidence relating cadmium exposure and cancer in humans has been found. The available epidemiological studies have examined relatively small numbers of workers and no causal relationship has been established; the limited data, however, may indicate a possible relationship between cadmium exposure and cancer of the prostate.

Of eight deaths in a group of 74 workers with at least ten years of exposure to cadmium oxide, three resulted from cancer of the prostate and two from other cancers. The actual exposure levels were unknown but were sufficient to cause proteinuria and anosmia (loss of the sense of smell) in many workers (Potts, 1965). No control group was examined. Four cases of cancer of the prostate versus an expected number of 0.58 were seen in a group of 248 workers exposed to cadmium oxide for a minimum of one year (Kipling and Waterhouse, 1967).

Morgan (1970) found cadmium levels in liver and kidney to be significantly higher in persons with bronchogenic carcinoma than in controls (Table 6.10). Later, Morgan (1971) reanalyzed the 1970 data with respect to emphysema, lung cancer, lung cancer plus emphysema, and controls. Both lung cancer and emphysema were independently associated with excess cadmium. Cadmium levels in kidney and liver of persons with carcinoma of the lung, with or without emphysema, significantly exceeded those in controls. Liver, but not kidney, tissue contained significantly higher cadmium levels than controls in cases with pure emphysema (Table 6.11). Although liver and kidney cadmium levels were significantly higher than normal in persons with lung cancer, no causal relationship could be proven since trace-metal concentrations have been shown to be abnormal in association with many neoplastic diseases. In addition, both lung cancer and emphysema have been linked to cigarette smoking; the body burden of cadmium is higher in smokers than in nonsmokers (Friberg et al., 1974).

TABLE 6.10. CADMIUM LEVELS IN LIVER AND KIDNEY WITH RESPECT TO LUNG AND OTHER CANCERS

Condition	Number of persons	Cadmium concentration (ppm, ash)	
		Liver	Kidney
Lung cancer	47	254 ± 133	3513 ± 1587
Other cancer	50	179 ± 140	2937 ± 2065
Control	55	182 ± 99	2406 ± 1299

Source: Adapted from Morgan, 1970, Table 1, p. 1395. Reprinted by permission of the publisher.

TABLE 6.11. CADMIUM LEVELS IN LIVER AND KIDNEY WITH RESPECT TO LUNG CANCER AND/OR EMPHYSEMA

Condition	Number of persons	Cadmium concentration (ppm, ash)	
		Liver	Kidney
Lung cancer	23	249 ± 142[a]	3478 ± 1639[a]
Emphysema	26	290 ± 155[a]	2921 ± 1252
Lung cancer and emphysema	19	268 ± 43[a]	3660 ± 1602[a]
Control	36	170 ± 95	2512 ± 1427

[a] $P < 0.05$.

Source: Adapted from Morgan, Burch, and Watkins, 1971, Table 1, p. 109. Reprinted by permission of the publisher.

The concern that a causal relationship may exist between cadmium exposure and cancer in humans stems from the ability of cadmium to induce cancer in experimental animals. Subcutaneous or intramuscular injections of cadmium in rats have led to sarcomas at the injection site in several experiments. For instance, 14 of 20 rats given subcutaneous injections of cadmium sulfate developed injection site sarcomas; testicular site atrophy and Leydig cell hyperplasia and neoplasia were also found (Haddow et al., 1964; Roe et al., 1964). The same treatment did not produce tumors in mice. In another study, a single subcutaneous or intramuscular injection of 0.17 to 0.34 mg cadmium chloride induced injection site sarcomas in rats (Gunn, Gould, and Anderson, 1967). No tumors were produced in skin, liver, salivary glands, prostate, or kidney. Most cancers induced by subcutaneous or intramuscular injection of cadmium compounds arise in tissues of mesenchymal (intramuscular) origin and not in tissues of ectodermal (skin), endodermal (e.g., liver and salivary gland), or epithelial (e.g., kidney) origin. Oral cadmium was not shown to be carcinogenic to mice, although rats given oral cadmium had more tumors than did controls (Kanisawa and Schroeder, 1969).

6.3.2.4.5 Teratogenesis — Cadmium has been shown to be teratogenic in rats, hamsters, and mice. Ferm and Carpenter (1968) injected pregnant golden hamsters intravenously with 2 or 4 mg/kg cadmium sulfate ($3CdSO_4 \cdot 8H_2O$), 2 mg/kg zinc sulfate ($ZnSO_4 \cdot 7H_2O$), or a mixture of the two (2 mg/kg each) on the eighth day of gestation (Table 6.12). The cadmium-induced abnormal development in the hamster was specific, affecting facial formation. Unilateral and bilateral cleft lips and palates are usual when cadmium exposure occurs

TABLE 6.12. CADMIUM TERATOGENICITY IN HAMSTERS

Treatment	Number of mothers treated	Total embryos	Percentage of embryos resorbed	Percentage of nonabsorbed embryos abnormal
Cadmium, 2 mg/kg	20	248	13	66
Cadmium, 4 mg/kg	14	190	46	84
Zinc, 2 mg/kg	6	70	3	4
Cadmium and zinc, 2 mg/kg each simultaneously	10	120	5	2
Cadmium, 2 mg/kg, plus zinc, 2 mg/kg 15 min to 6 hr later	26	276	12	12

Source: Adapted from Ferm and Carpenter, 1968, Table 1, p. 430. Reprinted by permission of the publisher.

on the eighth day of gestation. Mulvihill, Gamm, and Ferm (1970) discussed facial formation in normal and cadmium-treated hamsters. Although closure of the palatine shelves occurs between 12 and 14 days of gestation (Mulvihill, Gamm, and Ferm, 1970), the critical time for cadmium-induced abnormalities of palatal development is the eighth day of gestation (Ferm, 1971). When cadmium is given early in the eighth day, facial abnormalities predominate, but when given later on the eighth day, rib and upper limb abnormalities appear and facial defects decrease. When cadmium is given on the ninth day, most defects are of ribs and limbs (Ferm, 1971).

Doses of 0.8 to 2.0 mg cadmium per kilogram body weight were teratogenic when given intraperitoneally (but not subcutaneously) to pregnant Wistar rats on day 9, 10, or 11 of gestation (Barr, 1973). When given on day 6, 7, 8, or 12, cadmium was not teratogenic. A variety of defects occured and there were some differences between the two stocks of rats used. The incidence of the various defects changed with day of cadmium administration.

Daily subcutaneous injections of cadmium chloride into rats (4 to 12 mg/kg body weight) from days 13 through 16 produced a dose-related rise in fetal death rate, a decrease in fetal weight, and an increase in rate of fetal anomalies (Chernoff, 1973). Wolkowski (1974) reported that cadmium crossed the placenta and resulted in increased fetal mortality in mice.

There is no direct evidence for a teratogenic effect of cadmium in humans, but birth weights of children of women (aged 18 to 48) working in cadmium industries were significantly lower than weights of children born to control women (Cvetkova, 1970, as cited in Friberg et al., 1974). Four of 27 children born to women working in a zinc smelter in the above study had signs of rickets and delayed development of teeth. No causal relationship could be proved. Friberg et al. (1974) suggested that the teratogenic effect of cadmium might be due to a secondary zinc deficiency in the fetus since more zinc is stored in the mother's liver and kidneys during cadmium exposure. Cadmium does not, however, prevent the placental transfer of zinc (Ferm and Hanlon, 1974).

6.3.2.4.6 Mutagenesis — Cadmium-induced mutagenesis has not been experimentally demonstrated; however, an increased number of chromosomal abnormalities were found in peripheral leukocytes from itai-itai patients (Shiraishi and Yosida, 1972), and chromosomal abnormalities were induced in cultured human leukocytes by cadmium sulfide at 0.062 μg/ml of culture medium (Shiraishi, Kurahashi, and Yosida, 1972). Since a sulfide control, however, was not included in this study, the cytogenetic effects observed cannot be unequivocally ascribed to cadmium. Deaven and Campbell (1976) have reported that exposure of cultured hamster ovary cells to 0.224 ppm cadmium as $CdCl_2$ results in multiple chromatid aberrations within 24 to 48 hr and in shattering of chromsomes after 72 hr.

6.3.2.4.7 Itai-itai (ouch-ouch) disease — Itai-itai disease in Japan is regarded by many workers as a classic example of chronic cadmium poisoning resulting from industrial contamination of the environment (Friberg et al., 1974; Hise and Fulkerson, 1973). Because of the large amount of information on the various aspects of itai-itai disease, only a summary is presented here and the reader is referred to Friberg et al. (1974) for further details.

Itai-itai disease has been described as the culmination of many of the toxic effects and interactions of cadmium with other factors discussed in this chapter. Primarily, the source of cadmium was contaminated drinking water and rice grown on land irrigated with contaminated water; the cadmium contamination came from mining activities. Most of the persons affected were women who had reached menopause and had given birth to several children. The disease probably represents renal tubular dysfunction plus osteomalacia (softening of bones with pain, tenderness, muscular weakness, and loss of appetite, resulting from a vitamin D or calcium deficiency), and osteoporosis. There is evidence that cadmium can reduce calcium absorption from the gut and its uptake into bones (Ando et al., 1977). Standard symptoms of itai-itai disease are listed in Table 6.13.

Although cadmium is thought to be a causal factor in this disease, apparently other factors such as calcium and vitamin D deficiency are very likely also involved. This fact presumably explains the observation that the patients were multiparous women over 45 years of age. The combination of mineral loss from bone in connection with pregnancy and lactation, insufficient sunshine resulting in a lack of vitamin D (the women screen themselves from the sun while working), and generally poor nutrition during and immediately after World War II possibly produced in those women a calcium and phosphorus balance especially susceptible to disturbance in the presence of cadmium-induced renal tubular damage.

It should be pointed out that some questions have been raised about the identification of itai-itai disease with cadmium intoxication (Takeuchi,

TABLE 6.13. SYMPTOMS OF ITAI-ITAI DISEASE

A. Subjective symptoms:
    1. Pain (lumbago, back pain, joint pain)
    2. Disturbance of gait (duck gait)

B. Physical examination:
    1. Pain by pressure
    2. "Dwarfism"
    3. Kyphosis
    4. Restriction of spinal movement

C. X-ray examination:
    1. Milkman's pseudofractures
    2. Fractures (including callus formation)
    3. Thinned bone cortex
    4. Decalcification
    5. Deformation
    6. Fishbone vertebrae
    7. Coxa vara

D. Urine analysis:
    1. Coinciding positive tests for protein and glucose
    2. Protein (+)
    3. Glucose (+)
    4. Decreased phosphorus to calcium ratio

E. Serum analysis:
    1. Increased alkaline phosphatase
    2. Decreased serum inorganic phosphate

Source: Adapted from Friberg et al., Cadmium in the Environment, 2nd ed., p. 146, (c) CRC Press, Inc., 1974. Used by permission of CRC Press, Inc.

1973); very high therapeutic doses of vitamin D may have contributed to the renal tubular damage seen in these patients. Murata et al. (1970) considered itai-itai disease to represent primarily a cadmium enteropathy, which interferes with normal calcium absorption. Toxic effects of cadmium on the gastrointestinal tract have also been described by Valberg et al. (1977).

6.3.2.4.8 Effects on the nervous system — Like other heavy metals, cadmium can also interfere with activity of the nervous system. Friberg, Piscator, and Nordberg (1971) reported a variety of central effects in workers employed in a storage battery factory. Such symptoms may of course result indirectly from vascular effects of the metal. A direct action of cadmium on nerve cells has, however, also been noted (Gabbiani and Gregory, 1967; Kasuga, Sugawara, and Okada, 1974).

Several reports have appeared on effects of cadmium on synaptic transmission (Copper et al., 1975; Smirnov, Byzov, and Rampan, 1954). The action of the metal has been correlated with competitive inhibition of calcium movement at the presynaptic nerve terminal; this effect prevents normal release of acetylcholine following stimulation of the presynaptic nerve axon (Kober, 1977; Kober and Cooper, unpublished).

SECTION 6

REFERENCES

1. Adalis, D., D. E. Gardner, F. J. Miller, and D. L. Coffin. 1977. Toxic Effects of Cadmium on Ciliary Activity Using a Tracheal Ring Model System. Environ. Res. 13:111-120.

2. Adams, R. G., J. F. Harrison, and P. Scott. 1969. The Development of Cadmiun-Induced Proteinuria, Impaired Renal Function, and Osteomalacia in Alkaline Battery Workers. Q. J. Med. (Great Britain) 38(152):425-443.

3. Ahlmark, A., B. Axelsson, L. Friberg, and M. Piscator. 1961. Further Investigations into Kidney Function and Proteinuria in Chronic Cadmium Poisoning. Proc. Int. Congr. Occup. Health 13:201-203.

4. Ando, M., Y. Sayato, M. Tonomura, and T. Osawa. 1977. Studies on Excretion and Uptake of Calcium by Rats after Continuous Oral Administration of Cadmium. Toxicol. Appl. Pharmacol. 39:321-327.

5. Aschbacher, P. W. 1973. Air Pollution Research Needs: Livestock Production Systems. J. Air Pollut. Control Assoc. 23(4):267-272.

6. Axelsson, B., and M. Piscator. 1966. Serum Proteins in Cadmium Poisoned Rabbits. Arch. Environ. Health 12:374-381.

7. Baader, E. W. 1952. Chronic Cadmium Poisoning. Ind. Med. Surg. 21(9):427-430.

8. Barr, M., Jr. 1973. The Teratogenicity of Cadmium Chloride in Two Stocks of Wistar Rats. Teratology 7(3):237-242.

9. Barrett, H. M., and B. Y. Card. 1947. Studies on the Toxicity of Inhaled Cadmium: II. The Acute Lethal Dose of Cadmium Oxide for Man. J. Ind. Hyg. Toxicol. 29(5):286-293.

10. Berlin, M., and M. Piscator. 1961. Blood Volume in Normal and Cadmium-Poisoned Rabbits. Arch. Environ. Health 2:576-583.

11. Berlin, M., and S. Ullberg. 1963. The Fate of Cd^{109} in the Mouse. Arch. Environ. Health 7:686-688.

12. Berry, J. W., D. W. Osgood, and P. A. St. John. 1974. Chemical Villains — A Biology of Pollution. C. V. Mosby Company, St. Louis, Mo. 189 pp.

13. Beton, D. C., G. S. Andrews, H. J. Davies, L. Howells, and G. F. Smith. 1966. Acute Cadmium Fume Poisoning. Br. J. Ind. Med. (Great Britain) 23:292-301.

14. Bierenbaum, M. L. 1975. Possible Toxic Water Factor in Coronary Heart-Disease. Lancet (Great Britain) 2:76.

15. Bierenbaum, M. L., A. I. Fleischman, J. Dunn, and J. Arnold. 1975. Possible Toxic Water Factor in Coronary Heart-Disease. Lancet (Great Britain) 1:1008-1010.

16. Blejer, H. P., P. E. Caplan, and A. E. Alcocer. 1966. Acute Cadmium Fume Poisoning in Welders — A Fatal and a Nonfatal Case in California. Calif. Med. 105(4):290-296.

17. Bonnell, J. A. 1955. Emphysema and Proteinuria in Men Casting Copper-Cadmium Alloys. Br. J. Ind. Med. (Great Britain) 12:181-197.

18. Brisson, G. R., F. Malaisse-Lagae, and W. J. Malaisse. 1972. The Stimulus-Secretion Coupling of Glucose-Induced Insulin Release. J. Clin. Invest. 51:232-241.

19. Bruhn, J. C., and A. A. Franke. 1974. Lead and Cadmium in California Milk. J. Dairy Sci. 57:588.

20. Bunn, C. R., and G. Matrone. 1966. In Vivo Interactions of Cadmium, Copper, Zinc and Iron in the Mouse and Rat. J. Nutr. 90:395-399.

21. Burch, G. E., and J. J. Walsh. 1959. The Excretion and Biologic Decay Rates of Cd^{115m} with a Consideration of Space, Mass, and Distribution in Dogs. J. Lab. Clin. Med. 54(1):66-72.

22. Butt, E. M., R. E. Nusbaum, T. C. Gilmour, S. L. Didio, and Sister Mariano. 1964. Trace Levels in Human Serum and Blood. Arch. Environ. Health 8:52-57.

23. Carroll, R. E. 1966. The Relationship of Cadmium in the Air to Cardiovascular Disease Death Rates. J. Am. Med. Assoc. 198(3):177-179.

24. Caujolle, F., P. H. Chanh, G. Mamy, L. Suong, and J. Patte. 1964. Activité Comparée des Sulfates de Zinc et de Cadmium sur la Glycémie et la Cholestérolémie du Chien Chloralosé (Comparative Action of Zinc Sulfate and Cadmium on Glycemia and Cholesterolemia in the Chloralose Dog). Agressologie (France) 3:263-268.

25. Caujolle, F., J. Oustrin, and G. Silve-Mamy. 1971. Fixation et Circulation Entérophépatique du Cadmium. J. Eur. Toxicol. (France) 4:310-315.

26. Chatterjee, G. C., S. K. Banerjee, and D. Rudra Pal. 1973. Cadmium Administration and L-Ascorbic Acid Metabolism in Rats: Effect of L-Ascorbic Acid Supplementation. Int. J. Vitam. Nutr. Res. (Switzerland) 43:370-377.

27. Chaube, S., H. Nishimura, and C. A. Swinyard. 1973. Zinc and Cadmium in Normal Human Embryos and Fetuses. Arch. Environ. Health 26(5):237-240.

28. Chen, R. W., P. D. Whanger, and P. H. Weswig. 1975. Selenium-Induced Redistribution of Cadmium Binding to Tissue Proteins: A Possible Mechanism of Protection against Cadmium Toxicity. Bioinorg. Chem. 4:125-133.

29. Cherian, M. G., R. A. Goyer, and L. Delaquerriere-Richardson. 1976. Cadmium Metallothionein Induced Nephropathy. Toxicol. Appl. Pharmacol. 38:399.

30. Chernoff, N. 1973. Teratogenic Effects of Cadmium in Rats. Teratology 8(1):29-32.

31. Choudhury, H., L. Hastings, G. P. Cooper, and H. G. Petering. 1977. Dietary Cadmium: Embrytoxicity and Neonatal Behavioral Effects, Annual Report, Center for the Study of the Human Environment. Department of Environmental Health, University of Cincinnati, Cincinnati, Ohio. pp. 60-63.

32. Cooper, G. P., D. Fretthold, S. Shahbabian, L. J. Galea, and D. Steinberg. 1975. Lead and Cadmium Block Adrenergic Synaptic Transmission at the Rabbit Sympathetic Nerve—Arterial Muscle Junction. Physiologist 18:174.

33. Cornell, D. G., and M. J. Pallansch. 1973. Cadmium Analysis of Dried Milk by Pulse Polarographic Techniques. J. Dairy Sci. 56(12):1479-1485.

34. Crawford, M. D., M. J. Gardner, and J. N. Morris. 1968. Mortality and Hardness of Local Water-Supplies. Lancet (Great Britain) 1:827-831.

35. Deaven, L. L., and E. W. Campbell. 1976. Effects of Cadmium on Karyotype Stability in Chinese Hamster Ovary Cells; Progress Report January 1-June 30, 1976. U.S. National Technical Information Service Report LA-6451-PR. 4 pp.

36. Decker, C. F., R. U. Byerrum, and C. A. Hoppert. 1957. A Study of the Distribution and Retention of Cadmium-115 in the Albino Rat. Arch. Biochem. Biophys. 66:140-145.

37. Decker, L. E., R. U. Byerrum, C. F. Decker, C. A. Hoppert, and R. F. Langham. 1958. Chronic Toxicity Studies: I. Cadmium Administered in Drinking Water to Rats. AMA Arch. Ind. Health 18:228-231.

38. Dorn, C. R., J. O. Pierce II, G. R. Chase, and P. E. Phillips. 1973. Cadmium, Copper, Lead and Zinc in Blood, Milk, Muscle and Other Tissues of Cattle from an Area of Multiple-Source Contamination. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 7:191-203.

39. Duggan, R. E., and P. E. Corneliussen. 1972. Dietary Intake of Pesticide Chemicals in the United States (III), June 1968-April 1970. Pestic. Monit. J. 5:331-341.

40. Essing, H. G., K. H. Schaller, D. Szadkowski, and G. Lehnert. 1969. Usuelle Cadmiumbelastung durch Nahrungsmittel und Getränke (Usual Cadmium Loading through Foods and Drinks). Arch. Hyg. Bakteriol. (West Germany) 153(6):490-494.

41. Favino, A., F. Candura, G. Chiappino, and A. Cavalleri. 1968. Study on the Androgen Function of Men Exposed to Cadmium. Med. Lav. (Italy) 59(2):105-109.

42. Ferm, V. H. 1971. Developmental Malformations Induced by Cadmium. Biol. Neonate (Switzerland) 19:101-107.

43. Ferm, V. H., and S. J. Carpenter. 1968. The Relationship of Cadmium and Zinc in Experimental Mammalian Teratogenesis. Lab. Invest. 18(4):429-432.

44. Ferm, V. H., and D. P. Hanlon. 1974. Placental Transfer of Zinc in the Syrian Hamster during Early Embryogenesis. J. Reprod. Fertil. (Great Britain) 39:49-52.

45. Ferm, V. H., D. P. Hanlon, and J. Urban. 1969. The Permeability of the Hamster Placenta to Radioactive Cadmium. J. Embryol. Exp. Morphol. (Great Britain) 23(1):107-113.

46. Flick, D. F., H. F. Kraybill, and J. M. Dimitroff. 1971. Toxic Effects of Cadmium: A Review. Environ. Res. 4(2):71-85.

47. Forssen, A. 1972. Inorganic Elements in the Human Body: I. Occurrence of Ba, Br, Ca, Cd, Cs, Cu, K, Mn, Ni, Sn, Sr, Y and Zn in the Human Body. Ann. Med. Exp. Biol. Fenn. (Finland) 50:99-162.

48. Foulkes, E. C. 1974. Excretion and Retention of Cadmium, Zinc, and Mercury by the Rabbit Kidney. Am. J. Physiol. 227:1356.

49. Fox, M.R.S., and B. E. Fry, Jr. 1970. Cadmium Toxicity Decreased by Dietary Ascorbic Acid Supplements. Science 169:989-991.

50. Frant, S., and I. Kleeman. 1941. Cadmium "Food Poisoning." J. Am. Med. Assoc. 117(2):86-89.

51. Friberg, L. 1950. Health Hazards in the Manufacture of Alkaline Accumulators with Special Reference to Chronic Cadmium Poisoning. Acta. Med. Scand. Suppl. (Sweden) 138:63-100.

52. Friberg, L. 1952. Further Investigations on Chronic Cadmium Poisoning: A Study on Rabbits with Radioactive Cadmium. AMA Arch. Ind. Hyg. Occup. Med. 5:30-36.

53. Friberg, L., T. Kjellström, G. Nordberg, and M. Piscator. 1975. Cadmium in the Environment -- III. EPA-650/2-75-049, U.S. Environmental Protection Agency, Washington, D.C. 218 pp.

54. Friberg, L., M. Piscator, and G. Nordberg. 1971. Cadmium in the Environment — I. National Air Pollution Control Administration Publication APTD 0681.

55. Friberg, L., M. Piscator, G. F. Nordberg, and T. Kjellström. 1974. Cadmium in the Environment, 2nd ed. CRC Press, Cleveland, Ohio. 248 pp.

56. Fulkerson, W., and H. E. Goeller, eds. 1973. Cadmium, the Dissipated Element. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. 473 pp.

57. Gabbiani, G., D. Baic, and C. Déziel. 1967. Studies on Tolerance and Ionic Antagonism for Cadmium or Mercury. Can. J. Physiol. Pharmacol. (Canada) 45:443-450.

58. Gabbiani, G., and A. Gregory. 1967. Cadmium-Induced Selective Lesions of Sensory Ganglia. J. Neuropathol. Exp. Neurol. 26:498-506.

59. Ghafghazi, T., and J. H. Mennear. 1975. The Inhibitory Effect of Cadmium on the Secretory Activity of the Isolated Perfused Rat Pancreas. Toxicol. Appl. Pharmacol. 31:134-142.

60. Goodman, G. T., and T. M. Roberts. 1971. Plants and Soils as Indicators of Metals in the Air. Nature (Great Britain) 231:287-292.

61. Griffin, J. H., J. P. Rosenbusch, E. R. Blout, and K. K. Weber. 1973. Conformational Changes in Aspartate Transcarbamylase: II. Circular Dichroism Evidence for the Involvement of Metal Ions in Allosteric Interactions. J. Biol. Chem. 248(14):5057-5062.

62. Gunn, S. A., T. C. Gould, and W.A.D. Anderson. 1963. The Selective Injurious Response of Testicular and Epididymal Blood Vessels to Cadmium and Its Prevention by Zinc. Am. J. Pathol. 42(6):685-702.

63. Gunn, S. A., T. C. Gould, and W.A.D. Anderson. 1965. Strain Differences in Susceptibility of Mice and Rats to Cadmium-Induced Testicular Damage. J. Reprod. Fertil. (Great Britain) 10:273-275.

64. Gunn, S. A., T. C. Gould, and W.A.D. Anderson. 1966. Protective Effect of Thiol Compounds Against Cadmium-Induced Vascular Damage to Testis. Proc. Soc. Exp. Biol. Med. 122:1036-1039.

65. Gunn, S. A., T. C. Gould, and W.A.D. Anderson. 1967. Specific Response of Mesenchymal Tissue to Cancerigenesis by Cadmium. Arch. Pathol. 83:493-499.

66. Gunn, S. A., T. C. Gould, and W.A.D. Anderson. 1968. Mechanisms of Zinc, Cysteine and Selenium Protection against Cadmium-Induced Vascular Injury to Mouse Testis. J. Reprod. Fertil. (Great Britain) 15:65-70.

67. Haddow, A., F.J.C. Roe, C. E. Dukes, and B.C.V. Mitchley. 1964. Cadmium Neoplasia: Sarcomata at the Site of Injection of Cadmium Sulphate in Rats and Mice. Br. J. Cancer 18:667-673.

68. Hammer, D. I., J. F. Finklea, R. H. Hendricks, C. M. Shy, and R.J.M. Horton. 1971. Hair Trace Metal Levels and Environmental Exposure. Am. J. Epidemiol. 93(2):84-92.

69. Harrison, H. E., H. Bunting, N. K. Ordway, and W. S. Albrink. 1947. The Effects and Treatment of Inhalation of Cadmium Chloride in Aerosols in the Dog. J. Ind. Hyg. Toxicol. 29(5):302-314.

70. Hill, C. H., G. Matrone, W. L. Payne, and C. W. Barber. 1963. In Vivo Interactions of Cadmium with Copper, Zinc and Iron. J. Nutr. 80:227-235.

71. Hise, E. C., and W. Fulkerson. 1973. Environmental Impact of Cadmium Flow. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 203-322.

72. Hurt, W. F., Jr., C. Pinkerton, O. McNulty, and J. Creason. 1971. A Study in Trace Element Pollution of Air in 77 Midwestern Cities. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 4:56-68.

73. Ishizaki, A., M. Fukushima, and M. Sakamoto. 1970. On the Accumulation of Cadmium in the Bodies of Itai-Itai Patients. Jpn. J. Hyg. (Japan) 25:86.

74. Ishizu, S., M. Minami, A. Suzuki, M. Yamada, M. Sato, and K. Yamamura. 1973. An Experimental Study on Teratogenic Effect of Cadmium. Ind. Health (Japan) 11(3):127-139.

75. Ithakissios, D. S., T. Ghafghazi, J. H. Mennear, and W. V. Kessler. 1975. Effect of Multiple Doses of Cadmium on Glucose Metabolism and Insulin Secretion in the Rat. Toxicol. Appl. Pharmacol. 31:143-149.

76. Jacobs, E. E., M. Jacob, D. R. Sanadi, and L. B. Bradley. 1956. Uncoupling of Oxidative Phosphorylation by Cadmium Ion. J. Biol. Chem. 223:147-156.

77. Kanisawa, M., and H. A. Schroeder. 1969. Life Term Studies on the Effect of Trace Elements on Spontaneous Tumors in Mice and Rats. Cancer Res. 29:892-895.

78. Kasuga, M., N. Sugawara, and A. Okada. 1974. Toxic Effects of Cadmium Stearate on Rat Cerebellum in Culture. Bull. Environ. Contam. Toxicol. 12:535-540.

79. Kazantzis, G., F. V. Flynn, J. S. Spowage, and D. G. Trott. 1963. Renal Tubular Malfunction and Pulmonary Emphysema in Cadmium Pigment Workers. Q. J. Med. (Great Britain) 32(126):165-192.

80. Kimura, M., and N. Otaki. 1972. Percutaneous Absorption of Cadmium in Rabbit and Hairless Mouse. Ind. Health (Japan) 10(1-2):7-10.

81. Kipling, M. D., and J.A.H. Waterhouse. 1967. Cadmium and Prostatic Carcinoma. Lancet (Great Britain) 1:730-731.

82. Kober, T. 1977. The Mechanism of Action of Lead and Cadmium Block in the Bullfrog Sympathetic Ganglion. Ph.D. Dissertation. University of Cincinnati, Cincinnati, Ohio.

83. Kober, T., and G. P. Cooper. Personal Communication.

84. Lauwerys, R. R., J. P. Buchet, H. A. Roels, J. Browers, and D. Stanescu. 1974. Epidemiological Survey of Workers Exposed to Cadmium. Arch. Environ. Health 28:145-148.

85. Lener, J., and B. Bibr. 1970. Cadmium Content in Some Foodstuffs in Respect of Its Biological Effects. Vitalst. Zivilisationskr. (West Germany) 15:139-141.

86. Lewis, G. P., W. J. Jusko, and L. L. Coughlin, and S. Hartz. 1972*a*. Cadmium Accumulation in Man: Influence of Smoking, Occupation, Alcholic Habit and Disease. J. Chronic Dis. (Great Britain) 25:717-726.

87. Lewis, G. P., W. J. Jusko, L. L. Coughlin, and S. Hartz. 1972*b*. Contribution of Cigarette Smoking to Cadmium Accumulation in Man. Lancet (Great Britain) 1:291-292.

88. Lynch, G. P., D. G. Cornell, and D. F. Smith. 1974. Excretion of Cadmium and Lead into Milk. In: Trace Element Metabolism in Animals — 2, W. G. Hoekstra, J. W. Suttie, H. E. Ganther, and W. Mertz, eds. University Park Press, Baltimore, Md. pp. 470-472.

89. McCabe, L. J., J. M. Symons, R. D. Lee, and G. G. Roebeck. 1970. Survey of Community Water Supply Systems. J. Am. Water Works Assoc. 62:670-687.

90. McClanahan, B. J., R. O. McClellan, J. R. McKenney, and L. K. Bustad. 1965. Milk Secretion of Zinc and Cadmium in the Ruminant. In: Radioisotopes in Animal Nutrition and Physiology. International Atomic Energy Agency, Vienna. pp. 173-188.

91. Menden, E. E., V. J. Elia, L. W. Michael, and H. G. Petering. 1972. Distribution of Cadmium and Nickel of Tobacco during Cigarette Smoking. Environ. Sci. Technol. 6(9):830-832.

92. Miller, W. J., D. M. Blackmon, R. P. Gentry, and F. M. Pate. 1969. Effect of Dietary Cadmium on Tissue Distribution of ^{109}Cadmium following a Single Oral Dose in Young Goats. J. Dairy Sci. 52(12):2029-2035.

93. Miller, W. J., D. M. Blackmon, and Y. G. Martin. 1968. ^{109}Cadmium Absorption, Excretion, and Tissue Distribution following Single Tracer Oral and Intravenous Doses in Young Goats. J. Dairy Sci. 51(11):1836-1839.

94. Miller, W. J., B. Lampp, G. W. Powell, C. A. Salotti, and D. M. Blackmon. 1967. Influence of a High Level of Dietary Cadmium on Cadmium Content in Milk, Excretion, and Cow Performance. J. Dairy Sci. 50(9):1404-1408.

95. Ministry of Agriculture, Fisheries, and Food. 1973. Survey of Cadmium in Food, Fourth Report. Her Majesty's Stationery Office, London, England. 32 pp.

96. Molokhia, M. M., and H. Smith. 1967. Trace Elements in the Lung. Arch. Environ. Health 15:745-750.

97. Moore, W., Jr., J. F. Stara, and W. C. Crocker. 1973. Gastrointestinal Absorption of Different Compounds of ^{115m}Cadmium and the Effect of Different Concentrations in the Rat. Environ. Res. 6:159-164.

98. Moore, W., Jr., J. F. Stara, W. C. Crocker, M. Malanchuk, and R. Iltis. 1973. Comparison of ^{115m}Cadmium Retention in Rats following Routes of Administration. Environ. Res. 6:473-478.

99. Morgan, J. M. 1969. Tissue Cadmium Concentration in Man. Arch. Intern. Med. 123:405-408.

100. Morgan, J. M. 1970. Cadmium and Zinc Abnormalities in Bronchogenic Carcinoma. Cancer 25(6):1394-1398.

101. Morgan, J. M., H. B. Burch, and J. B. Watkins. 1971. Tissue Cadmium and Zinc Content in Emphysema and Bronchogenic Carcinoma. J. Chronic Dis. (Great Britain) 24:107-110.

102. Morris, J. N., M. D. Crawford, and J. A. Heady. 1961. Hardness of Local Water-Supplies and Mortality from Cardiovascular Disease. Lancet (Great Britain) 1:860-862.

103. Morton, W. E. 1971. Hypertension and Drinking Water Constituents in Colorado. Am. J. Public Health 61(7):1371-1378.

104. Mulvihill, J. E., S. H. Gamm, and V. H. Ferm. 1970. Facial Formation in Normal and Cadmium-Treated Golden Hamsters. J. Embryol. Exp. Morphol. (Great Britain) 24(2):393-403.

105. Munshower, F. F. 1972. Cadmium Compartmentation and Cycling in a Grassland Ecosystem in the Deer Lodge Valley, Montana. Ph.D. Dissertation. University of Montana, Missoula, Mont. 106 pp.

106. Murata, I., T. Hirono, V. Saeki, and S. Nakagawa. 1970. Cadmium Enteropathy, Renal Osteomalacia ("Itai-Itai Disease in Japan"). Bull. Soc. Int. Chir. (Belgium) 29:34-42.

107. Murthy, G. K. 1974. Trace Elements in Milk. CRC Critical Reviews in Environmental Control. CRC Press, Cleveland, Ohio. p. 5.

108. Murthy, G. K., and U. Rhea. 1968. Cadmium and Silver Content of Market Milk. J. Dairy Sci. 51(4):610-613.

109. Mustafa, M. G., C. E. Cross, and W. S. Tyler. 1971. Interference of Cadmium Ion with Oxidative Metabolism of Alveolar Macrophages. Arch. Intern. Med. 127:1050-1058.

110. Mustafa, M. G., P. A. Peterson, R. J. Munn, and C. E. Cross. 1971. Effects of Cadmium Ion on Metabolism of Lung Cells. Proc. Int. Clean Air Congr. 2:143-151.

111. National Institute of Occupational Safety and Health. 1976. Criteria for a Recommended Standard — Occupational Exposure to Cadmium. HEW Publication No. (NIOSH) 76-192.

112. Natusch, D.F.S., J. R. Wallace, and C. A. Evans, Jr. 1974. Toxic Trace Elements: Preferential Concentration in Respirable Particles. Science 183:202-204.

113. Nomiyama, K., and E. C. Foulkes. 1977. Renal Reabsorption of Filtered Cadmium-Metallothionein in the Rabbit Kidney. Proc. Soc. Exp. Biol. Med. (in press).

114. Nomiyama, K., H. Nomiyama, M. Yotoriyama, and T. Taguchi. 1977. Some Recent Studies on the Renal Effects of Cadmium (presented at the First International Cadmium Conference, San Francisco, February 1977).

115. Nordberg, G. F. 1971. Effects of Acute and Chronic Cadmium Exposure on the Testicles of Mice. Environ. Physiol. (Denmark) 1:171-187.

116. Nordberg, G. F. 1972. Cadmium Metabolism and Toxicity. Environ. Physiol. Biochem. (Denmark) 2:7-36.

117. Nordberg, G. F. 1974. Health Hazards of Environmental Cadmium Pollution. Ambio (Norway) 3(2):55-66.

118. Nordberg, G. F., R. Goyer, and M. Nordberg. 1975. Comparative Toxicity of Cadmium-Metallothionein and Cadmium Chloride on Mouse Kidney. Arch. Pathol. 99:192-197.

119. Norton, K. B., and J. E. Kench. 1977. Effects of Cadmium on Ribosomal Protein Synthesis in Rat Liver. Environ. Res. 13:102-110.

120. Parizek, J. 1957. The Destructive Effect of Cadmium Ion on Testicular Tissue and Its Prevention by Zinc. J. Endocrinol. (Great Britain) 15:56-63.

121. Parizek, J. 1964. Vascular Changes at Sites of Oestrogen Biosynthesis Produced by Parenteral Injection of Cadmium Salts: The Destruction of Placenta by Cadmium Salts. J. Reprod. Fertil. (Great Britain) 7:263-265.

122. Parizek, J. 1965. The Peculiar Toxicity of Cadmium during Pregnancy — An Experimental Toxaemia of Pregnancy Induced by Cadmium Salts. J. Reprod. Fertil. (Great Britain) 9:111-112.

123. Parizek, J., and Z. Zahor. 1956. Effect of Cadmium Salts on Testicular Tissue. Nature (Great Britain) 177:1036-1037.

124. Paterson, J. C. 1947. Studies on the Toxicity of Inhaled Cadmium: III. The Pathology of Cadmium Smoke Poisoning in Man and in Experimental Animals. J. Ind. Hyg. Toxicol. 29(5):294-301.

125. Perry, H. M., Jr., and M. W. Erlanger. 1973. Elevated Circulating Renin Activity in Rats following Doses of Cadmium Known to Induce Hypertension. J. Lab. Clin. Med. 82(3):399-405.

126. Perry, H. M., M. Erlanger, A. Yunice, E. Schoepfle, and E. F. Perry. 1970. Hypertension and Tissue Metal Levels following Intravenous Cadmium, Mercury, and Zinc. Am. J. Physiol. 219(3):755-761.

127. Pinkerton, C., D. I. Hammer, K. Bridbord, J. P. Creason, J. L. Kent, and G. K. Murthy. 1973. Human Milk as a Dietary Source of Cadmium and Lead. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 6:39-43.

128. Piscator, M. 1966. Proteinuria in Chronic Cadmium Poisoning: III. Electrophoretic and Immunoelectrophoretic Studies on Urinary Proteins from Cadmium Workers, with Special Reference to the Excretion of Low Molecular Weight Proteins. Arch. Environ. Health 12:335-344.

129. Piscator, M., and B. Axelsson. 1970. Serum Proteins and Kidney Function after Exposure to Cadmium. Arch. Environ. Health 21:604-608.

130. Pond, W. G., and E. F. Walker, Jr. 1972. Cadmium-Induced Anemia in Growing Rats: Prevention by Oral or Parenteral Iron. Nutr. Rep. Int. 5(6):365-370.

131. Pond, W. G., E. F. Walker, Jr., and D. Kirtland. 1973. Cadmium-Induced Anemia in Growing Pigs: Protective Effect of Oral or Parenteral Iron. J. Anim. Sci. 36(6):1122-1124.

132. Porter, M. C., T. S. Miya, and W. F. Bousquet. 1974. Cadmium: Inability to Induce Hypertension in the Rat. Toxicol. Appl. Pharmacol. 27:692-695.

133. Potts, C. L. 1965. Cadmium Proteinuria — The Health of Battery Workers Exposed to Cadmium Oxide Dust. Ann. Occup. Hyg. (Great Britain) 8:55-61.

134. Pribble, H. J., and P. H. Weswig. 1973. Effects of Aqueous and Dietary Cadmium on Rat Growth and Tissue Uptake. Bull. Environ. Contam. Toxicol. 9(5):271-275.

135. Princi, F. 1947. A Study of Industrial Exposures to Cadmium. J. Ind. Hyg. Toxicol. 29(5):315-320.

136. Rahola, T., R. K. Aaran, and J. K. Miettinen. 1972. Half-Time Studies of Mercury and Cadmium by Whole-Body Counting. In: Assessment of Radioactive Contamination in Man. International Atomic Energy Agency, Vienna. pp. 553-562.

137. Roe, F.J.C., C. E. Dukes, K. M. Cameron, R.C.B. Pugh, and B.C.V. Mitchley. 1964. Cadmium Neoplasia: Testicular Atrophy and Leydig Cell Hyperplasia and Neoplasia in Rats and Mice following the Subcutaneous Injection of Cadmium Salts. Br. J. Cancer (Great Britain) 18:674-681.

138. Sabbioni, E., and E. Marafante. 1975. Heavy Metals in Rat Liver Cadmium Binding Protein. Environ. Physiol. Biochem. (Denmark) 5:132-141.

139. Sansi, K.A.O., and W. G. Pond. 1974. Pathology of Dietary Cadmium Toxicity in Growing Rats and the Protective Effect of Injected Iron. Nutr. Rep. Int. 9(6):407-414.

140. Schroeder, H. A. 1964. Cadmium Hypertension in Rats. Am. J. Physiol. 207:62-66.

141. Schroeder, H. A. 1965. Cadmium as a Factor in Hypertension. J. Chronic Dis. (Great Britain) 18:647-656.

142. Schroeder, H. A., and J. J. Balassa. 1961. Abnormal Trace Metals in Man: Cadmium. J. Chronic Dis. (Great Britain) 14(2):236-258.

143. Schroeder, H. A., and J. Buckman. 1967. Cadmium Hypertension. Arch. Environ. Health 14:693-697.

144. Schroeder, H. A., and A. P. Nason. 1969. Trace Metals in Human Hair. J. Invest. Dermatol. 53(1):71-78.

145. Schroeder, H. A., A. P. Nason, and J. J. Balassa. 1967. Trace Metals in Rat Tissues as Influenced by Calcium in Water. J. Nutr. 93:331-336.

146. Schroeder, H. A., A. P. Nason, and M. Mitchener. 1968. Action of a Chelate of Zinc on Trace Metals in Hypertensive Rats. Am. J. Physiol. 214(4):796-800.

147. Schroeder, H. A., A. P. Nason, I. H. Tipton, and J. J. Balassa. 1967. Essential Trace Metals in Man: Zinc, Relation to Environmental Cadmium. J. Chronic Dis. (Great Britain) 20:179-210.

148. Sharrett, A. R., and M. Feinleib. 1975. Possible Toxic Water Factor in Coronary Heart-Disease. Lancet (Great Britain) 2:76.

149. Shiraishi, Y., H. Kurahashi, and T. H. Yosida. 1972. Chromosomal Aberrations in Cultured Human Leucocytes Induced by Cadmium Sulfide. Proc. Jpn. Acad. (Japan) 48(2):133-137.

150. Shiraishi, Y., and T. H. Yosida. 1972. Chromosomal Abnormalities in Cultured Leucocyte Cells from Itai-Itai Disease Patients. Proc. Jpn. Acad. (Japan) 48(4):248-251.

151. Singhal, R. L., Z. Merali, S. Kacew, and D.J.B. Sutherland. 1974. Persistence of Cadmium-Induced Metabolic Changes in Liver and Kidney. Science 183:1094-1096.

152. Skog, E., and J. E. Wahlberg. 1964. A Comparative Investigation of the Percutaneous Absorption of Metal Compounds in the Guinea Pig by Means of the Radioactive Isotopes: ^{51}Cr, ^{58}Co, ^{65}Zn, ^{110m}Ag, ^{115m}Cd, ^{203}Hg. J. Invest. Dermatol. 43:187-192.

153. Smirnov, G. D., A. L. Byzov, and I. I. Rampan. 1954. Role of Tissue Sulfhydryl Groups on Secretion of Acetylcholine and on Transmission of Stimuli in the Superior Cervical Ganglion in Cat. Friziol. Zh. SSSR im. I. M. Sechenova (U.S.S.R.) 40:424-430.

154. Smith, J. C., and J. E. Kench. 1957. Observations on Urinary Cadmium and Protein Excretion in Men Exposed to Cadmium Oxide Dust and Fume. Br. J. Ind. Med. (Great Britain) 14:240-245.

155. Smith, R. H. 1973. Mechanisms of Biological Action. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 273-278.

156. Smith, R. H., and J. W. Huckabee. 1973. Ecological Studies of the Movement, Fate, and Consequences of Cadmium. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 278-322.

157. Stowe, H. D., M. Wilson, and R. A. Goyer. 1972. Clinical and Morphologic Effects of Oral Cadmium Toxicity in Rabbits. Arch. Pathol. 94:389-405.

158. Suda, T., N. Horiuchi, E. Ogata, I. Ezawa, N. Otaki, and M. Kimura. 1974. Prevention by Metallothionein of Cadmium-Induced Inhibition of Vitamin D Activation Reaction of Kidney. FEBS Lett. (Netherlands) 42:23.

159. Suzuki, S., T. Suzuki, and M. Ashizawa. 1965. Proteinuria Due to Inhalation of Cadmium Stearate Dust. Ind. Health (Japan) 3:73-85.

160. Suzuki, S., and T. Taguchi. 1970. Sex Difference of Cadmium Content in Spot Urines. Ind. Health (Japan) 8:150-152.

161. Syversen, T.L.M. 1975. Cadmium-Binding in Human Liver and Kidney. Arch. Environ. Health 30:158-161.

162. Szadkowski, V. D., K. H. Schaller, and G. Lehnert. 1969. Renale Cadmiumausscheidung, Lebensalter und arterieller Blutdruck (The Relationship between Renal Cadmium Excretion, Age, and Arterial Blood Pressure). Z. Klin. Chem. Klin. Biochem. (West Germany) 7:551-552.

163. Takeuchi, J. 1973. Ethiology of Itai-itai Disease: Criticism on the Theory of Cadmium Source. Jpn. J. Clin. Med. 31:2048-2057.

164. Tanaka, M., N. Matsusaka, A. Yuyama, and H. Kobayashi. 1972. Transfer of Cadmium through Placenta and Milk in the Mouse. Radioisotopes (Japan) 21(1):50-52.

165. Task Group on Lung Dynamics. 1966. Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract. Health Phys. (Great Britain) 12:173-207.

166. Task Group on Metal Accumulation. 1973. Accumulation of Toxic Metals with Special Reference to Their Absorption, Excretion and Biological Half-Times. Environ. Physiol. Biochem. (Denmark) 3:65-107.

167. Terhaar, C. J., E. Vis, R. L. Roudabush, and D. W. Fassett. 1965. Protective Effects of Low Doses of Cadmium Chloride against Subsequent High Oral Doses in the Rat (abstract). Toxicol. Appl. Pharmacol. 7:500.

168. Thind, G. S., G. Karreman, K. F. Stephan, and W. S. Blakemore. 1970. Vascular Reactivity and Mechanical Properties of Normal and Cadmium-Hypertensive Rabbits. J. Lab. Clin. Med. 76(4):560-568.

169. Tipton, I. H., and M. J. Cook. 1963. Trace Elements in Human Tissue: II. Adult Subjects from the United States. Health Phys. (Great Britain) 9:103-145.

170. Tipton, I. H., H. A. Schroeder, H. M. Perry, and M. J. Cook. 1965. Trace Elements in Human Tissue: III. Subjects from Africa, the Near East and Far East and Europe. Health Phys. (Great Britain) 11:403-451.

171. Tipton, I. H., and J. J. Shafer. 1964. Statistical Analysis of Lung Trace Element Levels. Arch. Environ. Health 8:58-67.

172. Tipton, I. H., and P. L. Stewart. 1969. Patterns of Elemental Excretion in Long-Term Balance Studies. II. In: Health Physics Division Annual Progress Report for Period Ending July 31, 1969. ORNL-4446, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 303-304.

173. Tipton, I. H., P. L. Stewart, and J. Dickson. 1969. Patterns of Elemental Excretion in Long Term Balance Studies. Health Phys. (Great Britain) 16:455-462.

174. Tsuchiya, K. 1967. Proteinuria of Workers Exposed to Cadmium Fume. Arch. Environ. Health 14:875-880.

175. U.S. Department of Health, Education, and Welfare. 1962. Public Health Service Drinking Water Standards, Washington, D.C. 61 pp.

176. U.S. Department of Health, Education, and Welfare. 1969. Air Quality Criteria for Particulate Matter. Washington, D.C. 211 pp.

177. U.S. Environmental Protection Agency. 1972. Helena Valley Montana, Area Environmental Pollution Study. Office of Air Programs Publication No. AP-91, Research Triangle Park, N.C. 194 pp.

178. Vahlberg, L. S., J. Haist, M. G. Cherian, L. Delaquerriere-Richardson, and R. A. Goyer. 1977. Cadmium-Induced Enteropathy: Comparative Toxicity of Cadmium Chloride and Cadmium-Thionein. J. Toxicol. Environ. Health 2:963-975.

179. Vinegar, A., and H. Choudhury. 1976. Effects of Cadmium in Drinking Water on Pulmonary Function of Rats. In: Annual Report, Center for the Study of the Human Environment. Department of Environmental Health, University of Cincinnati, Cincinnati, Ohio. pp. 124-125.

180. Voinar, A. O. 1952. The Existence of and the Role of Cadmium in the Organism of Animals and Man. In: Mikroelementy v. Zhizni Rastenii i Zhivotnykh, Akad. Nauk S.S.S.R., Trudy Knof. Mikroelement, 1950, Moscow. pp. 55-70.

181. Voors, A. W., M. S. Shuman, and P. N. Gallagher. 1973. Zinc and Cadmium Autopsy Levels for Cardiovascular Disease in Geographical Context. Proc. Univ. Mo. Annu. Conf. Environ. Health 6:215-222.

182. Walsh, J. J., and G. E. Burch. 1959. The Rate of Disappearance from Plasma and Subsequent Distribution of Radiocadmium (Cd^{115m}) in Normal Dogs. J. Lab. Clin. Med. 54:59-65.

183. Webb, M. 1972. Protection by Zinc against Cadmium Toxicity. Biochem. Pharmacol. (Great Britain) 21:2767-2771.

184. Webb, M. 1975. Toxicity of Cadmium-Metallothionein (abstract). International Conference on Heavy Metals in the Environment, Toronto.

185. Wilson, R. H., F. DeEds, and A. J. Cox, Jr. 1941. Effects of Continued Cadmium Feeding. J. Pharmacol. Exp. Ther. 71:222-235.

186. Wolkowski, R. M. 1974. Differential Cadmium-Induced Embryotoxicity in Two Inbred Mouse Strains: I. Analysis of Inheritance of the Response to Cadmium and of the Presence of Cadmium in Fetal and Placental Tissues. Teratology 10(3):243-262.

187. Worker, N. A., and B. B. Migicovsky. 1961. Effect of Vitamin D on the Utilization of Zinc, Cadmium and Mercury in the Chick. J. Nutr. 74:222-224.

SECTION 7

ENVIRONMENTAL DISTRIBUTION AND TRANSFORMATION

7.1 SUMMARY

Cadmium is a relatively rare metal usually present in small amounts in zinc ores and commercially obtained as a by-product from the zinc-, copper-, and lead-producing industries. Its major uses are in electroplating, in pigment production, in plastic stabilizers, and in batteries. Little cadmium is recovered from spent products, and the supply-demand-cost relationships of cadmium production are directly related to the demand-cost of zinc production.

Sources of cadmium pollution include smelter fumes and dust, incineration of cadmium-bearing materials, burning of fossil fuels, fertilizer application, industrial waste disposal into water systems, and municipal wastewater discharge and sludge disposal. Thus, the highest concentrations of cadmium are most likely to be in the localized regions of smelters or in industrialized urban areas.

Background levels of atmospheric cadmium are typically quite low and often are below the limits of detection. Contents in urban areas are variable; reported cadmium levels range from 0 to as high as 0.150 $\mu g/m^3$, with means usually near 0.005 to 0.015 $\mu g/m^3$.

Uncontaminated soil contains 0.4 ppm cadmium or less. Soil in the vicinity of smelters contains high levels of cadmium (about 10 to 100 ppm), which typically decrease with distance from the smelter and with soil depth; soil in industrialized urban areas contains about 0.1 to 1 ppm cadmium.

Natural waters also contain highly variable amounts of cadmium; waters near industrial areas have values from 1 to 100 ppb, whereas many open rural waters contain less than 1 ppb. Sediments also contain highly variable amounts of cadmium, depending upon proximity to pollution outfalls.

Various processes serve to move cadmium from one medium to another. Precipitation and fallout deposit airborne cadmium on land or water. Leaching of soil can extract cadmium; the amount removed depends on pH, the chemical form of cadmium, and the type of soil. Runoff can carry particulate cadmium into surface waters. The fate of cadmium in waters depends on several factors; however, much of the cadmium is eventually deposited in sediments.

Because cadmium is present in a variety of industrial and municipal wastewaters and sludges, methods of water treatment are needed to reduce the cadmium content and methods of safe sludge disposal are necessary to reduce the possible release of toxic heavy metals into the environment.

7.2 TRENDS IN PRODUCTION AND USAGE

Cadmium is a relatively rare metal in the earth's crust; the most common geologic form, greenockite (CdS), occurs in a commercially important zinc ore, sphalerite (ZnS). Table 7.1 gives data on the natural abundance of zinc and cadmium in the environment (Goeller, Hise, and Flora, 1973).

TABLE 7.1. NATURAL ABUNDANCE OF ZINC AND CADMIUM

Source	Abundance (ppm) Zn	Abundance (ppm) Cd	Relative abundance of Zn[a]	Cd/Zn (%)
Worldwide	80	0.18	1.46	0.23
Continental	55	0.15 0.5	1.00	0.27
Ultramafic rocks	150	0.03	2.73	
Basaltic rocks	112	0.22	2.04	0.20
High-calcium granites	47	0.13	0.85	0.28
Low-calcium granites	39	0.13	0.71	0.33
Syenitic rocks	26	0.13	0.47	0.50
Average for igneous rocks	70	0.2	1.27	0.27
Shales	45-95	0.3	0.82-1.72	0.67-0.32
Sandstone	16	0.05	0.29	0.31
Limestone	20	0.035	0.36	0.17
Soil				
Range	10-300	0.01-0.7	0.2-5.4	0.1-0.23
Average	50	0.06	0.9	0.12
Seawater	10 ppb	0.1 ppb		1.0

[a] Abundance for specific rock type/continental abundance.

Source: Adapted from Goeller, Hise, and Flora, 1973, Table IV-1, p. 64.

The major commercial supply of cadmium is as a by-product in the production of zinc from sphalerite; smaller amounts are supplied as by-products from the copper- and lead-smelting industries. The processes involved in the extraction of zinc and cadmium for conversion to usable forms are mining, ore beneficiation, ore roasting and sintering, zinc smelting, cadmium recovery, and processing. Most of the cadmium is recovered from the ore roasting and sintering flue dusts by cadmium oxide reduction and from cadmium sulfate solutions (obtained from the purification of zinc) by electrolysis. Figure 7.1 represents a schematic flowsheet for cadmium recovery.

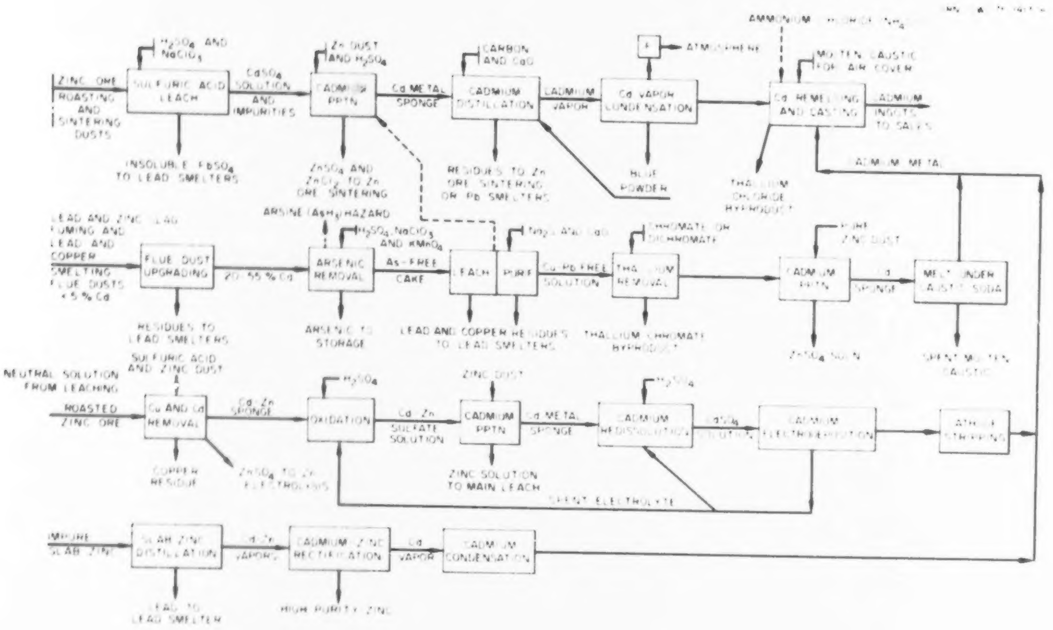

AMMONIUM CHLORIDE NH_4
H_2SO_4 AND $NaClO_3$
ZINC ORE ROASTING AND SINTERING DUSTS
SULFURIC ACID LEACH
$CdSO_4$ SOLUTION AND IMPURITIES
INSOLUBLE $PbSO_4$ TO LEAD SMELTERS
Zn DUST AND H_2SO_4
CADMIUM PPTN
Cd METAL SPONGE
$ZnSO_4$ AND $ZnCl_2$ TO Zn ORE SINTERING
CARBON AND CaO
CADMIUM DISTILLATION
RESIDUES TO Zn ORE SINTERING OR Pb SMELTERS
CADMIUM VAPOR
ATMOSPHERE
Cd VAPOR CONDENSATION
BLUE POWDER
MOLTEN CAUSTIC FOR AIR COVER
Cd REMELTING AND CASTING
CADMIUM INGOTS TO SALES
THALLIUM CHLORIDE BYPRODUCT
CADMIUM METAL
LEAD AND ZINC SLAG FUMING AND LEAD AND COPPER SMELTING FLUE DUSTS < 5 % Cd
FLUE DUST UPGRADING
20-55 % Cd
RESIDUES TO LEAD SMELTERS
ARSINE (AsH_3) HAZARD
H_2SO_4, $NaClO_3$ AND $KMnO_4$
ARSENIC REMOVAL
ARSENIC TO STORAGE
As-FREE CAKE
LEACH
PURIF
LEAD AND COPPER RESIDUES TO LEAD SMELTERS
Na_2S AND CaO
Cu-Pb FREE SOLUTION
CHROMATE OR DICHROMATE
THALLIUM REMOVAL
THALLIUM CHROMATE BYPRODUCT
PURE ZINC DUST
CADMIUM PPTN
$ZnSO_4$ SOL'N
Cd SPONGE
MELT UNDER CAUSTIC SODA
SPENT MOLTEN CAUSTIC
NEUTRAL SOLUTION FROM LEACHING
SULFURIC ACID AND ZINC DUST
ROASTED ZINC ORE
Cu AND Cd REMOVAL
COPPER RESIDUE
$ZnSO_4$ TO Zn ELECTROLYSIS
Cd-Zn SPONGE
H_2SO_4
OXIDATION
Cd-Zn SULFATE SOLUTION
ZINC DUST
CADMIUM PPTN
ZINC SOLUTION TO MAIN LEACH
Cd METAL SPONGE
H_2SO_4
CADMIUM REDISSOLUTION
$CdSO_4$ SOLUTION
CADMIUM ELECTRODEPOSITION
CATHODE STRIPPING
SPENT ELECTROLYTE
IMPURE SLAB ZINC
SLAB ZINC DISTILLATION
LEAD TO LEAD SMELTER
Cd-Zn VAPORS
CADMIUM-ZINC RECTIFICATION
HIGH PURITY ZINC
Cd VAPOR
CADMIUM CONDENSATION

Figure 7.1. Schematic flowsheet for recovery of cadmium as a by-product of zinc and lead recovery. Source: Goeller and Flora, 1973, Figure V-5, p. 148.

The major uses of cadmium are for electroplating, for pigments used in plastics, paints, enamels, and inks, for stabilizers in plastics, for alloys, and for nickel-cadmium batteries. Minor uses include electrical contact production, curing of rubber, use in fungicides, and use in solid-state systems (Heindl, 1970). Figure 7.2 shows a material flow diagram for cadmium in the United States and gives approximate amounts of cadmium used in each industry in 1968.

The determination of trends in production and use of cadmium is speculative and depends on many factors, including production and consumption of zinc, changing technology for recovery of cadmium from ores, development of substitutes for cadmium, and new uses for cadmium. Table 7.2 gives the percentage of total use of cadmium for each industry in 1968 and the projected use for the year 2000. The greatest projected increases are in electroplating of parts for motor vehicles, in plastics, and in batteries.

Because production of cadmium depends on zinc production, the output of cadmium is directly related to the demand for zinc and thus is related to the price of zinc. The projected world demand for zinc in the year 2000 lies between 6.1 x 10^9 kg and 11.5 x 10^9 kg (6,700,000 and 12,720,000 short tons). This amount would yield a cadmium supply between 2.45 x 10^7 kg and 4.63 x 10^7 kg (54,000,000 and 102,000,000 lb), which approximates the estimated use in the year 2000 — 2.3 x 10^7 to 3.6 x 10^7 kg (51,000,000 to

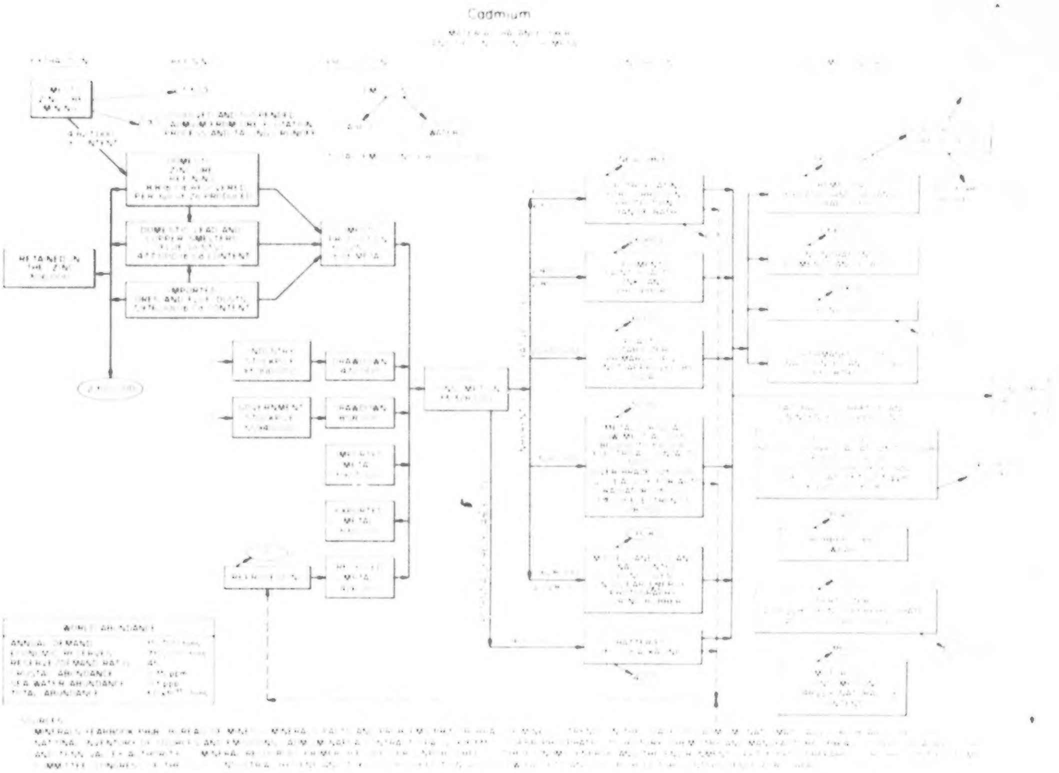


Figure 7.2. Societal flow of cadmium in the United States, 1968. Source: Goeller, Hise, and Flora, 1973, Figure IV-3, p. 86.

79,000,000 lb). Thus, if the high forecast for zinc demand is correct, there should be sufficient cadmium to meet projected demands; however, if the demand for zinc is lower, there will be insufficient cadmium to meet the estimated high demand. Because production of cadmium is insensitive to cadmium demand, some marginal uses of cadmium would become economically infeasible (Heindl, 1970).

An important problem arises from the fact that cadmium is used dispersively — little is recovered from discarded consumer or industrial products. With increased demand and improved technology, recovery techniques may be developed which can alter this supply-demand problem to a small extent. Presently, the recovery of cadmium from batteries seems the best possibility. Improvement in mine production technology may yield higher cadmium recovery from ores; however, this factor would be small since cadmium recovery from zinc ores is already estimated to be between 70% and 90% (Heindl, 1970). Goeller and Flora (1973) cited an example in which 98% of the cadmium was recovered; this value was obtained by adding the 82.5% recovered as marketable metal to the 15.9% produced as oxide residues.

TABLE 7.2. PROJECTED U.S. DEMAND FOR CADMIUM AND ZINC IN 2000 BASED ON USE IN 1968

Application	Use in 1968			Projected demand in 2000			Increase, 1968–2000 (%)	Change in fraction of total use (%)
	(metric tons)	(10^3 short tons)	(% of total)	(metric tons)	(10^3 short tons)	(% of total)		
			Cadmium					
Electroplating								
Motor vehicles	0.59	0.65	9.8	2.04	2.25	14.9	245	+52
Aircraft and boats	0.36	0.40	6.0	0.59	0.65	4.3	62	-38
Other	2.68	2.95	44.2	4.53	5.00	32.8	69	-26
Total electroplating	3.63	4.00	60.0	7.16	7.90	52.0	97	-13
Plastics	1.13	1.25	18.7	4.08	4.50	29.7	260	+59
Pigments	0.68	0.75	11.3	1.13	1.25	8.3	67	-27
Batteries	0.18	0.20	3.0	0.68	0.75	5.0	275	+67
Other	0.42	0.46	7.0	0.68	0.75	5.0	62	-29
Total	6.04	6.66	100.0	13.73	15.15	100.0	127	
			Zinc					
Construction	308	340	19.3	481	530	16.2	56	-16
Transportation	363	400	22.8	608	670	20.5	67	-10
Electrical equipment and supplies	190	210	11.9	662	730	22.4	247	+88
Plumbing and heating	217	240	13.6	363	400	12.2	67	-10
Other industrial machinery	145	160	9.1	236	260	8.0	62	-12
Pigments and compounds	200	220	12.5	336	370	11.3	68	-10
Rolled zinc	45	50	2.8	73	80	2.4	60	-16
Other	128	141	8.0	208	230	7.0	63	-13
Total	1596	1761	100.0	2967	3270	100.0	86	

Source: Adapted from Goeller, Hise, and Flora, 1973, Table IV-12, p. 104.

7.3 DISTRIBUTION OF CADMIUM IN THE ENVIRONMENT

Distribution of cadmium in the environment represents the resultant of natural occurrence and of cadmium added through natural redistributing processes and as a consequence of human activity.

7.3.1 Sources of Pollution

Total emissions of cadmium metal into air, soil, and water from all sources in the United States were estimated for 1968 to lie between 2.5 x 10^6 kg and 3.6 x 10^6 kg (5.5 and 7.9 million lb) (Goeller, Hise, and Flora, 1973). Data on amounts released at each stage in the material flow balance sheet are limited and should be determined to allow identification of crucial stages for pollution abatement priorities (Figure 7.2 and Table 7.3). The material balance sheet (Table 7.3) shows that while 2.5 to 3.6 x 10^6 kg (5 to 8 million lb) of cadmium are listed as emissions for 1968, an additional amount of almost 5 x 10^6 kg (11 million lb) is either delegated for permanent use or considered as waste with unknown disposition. Thus, total cadmium released to the environment each year may be considerably greater than the approximate value of 3.6 x 10^6 kg (8 million lb).

Major sources of cadmium pollution are processes involved in the extraction, refinement, and production of iron, steel, cadmium, zinc, lead, and copper. Other sources of cadmium pollution include the subsequent conversion of processed cadmium into various consumer products, emissions from use of fossil fuels, use of phosphate fertilizers with a high cadmium content, wearing of tires, and combustion of lubrication oil (Tables 7.3 and 7.4). Because cadmium content in fossil fuels and fertilizers varies with source, emission amounts can only be approximated (Goeller, Hise, and Flora, 1973). Data compiled by the MITRE Corporation (Table 7.4) show the amount of cadmium released from various industries and the percentage of the total that each source represents (Goldberg, 1973). Figure 7.3, using data from an independent study, lists emission amounts and flow of cadmium from various industries.

Differences exist among the estimates of cadmium emission into the environment. For example, Goldberg (1973) estimated the cadmium released by fertilizer application in the United States to be less than 907 kg (2000 lb), whereas Goeller, Hise, and Flora (1973) estimated it to lie between 2.3 x 10^4 and 2.3 x 10^5 kg (50,000 and 500,000 lb). This latter value seems more reasonable since the estimate for the amount of cadmium released by fertilizer application in Sweden was about 10,000 kg (22,050 lb) (Stenström and Vahter, 1974).

Cadmium pollution is associated with production, conversion, and refining industries. Figures 7.4 and 7.5 show major U.S. deposits of zinc and lead and the locations of major U.S. zinc production mines. Because cadmium is obtained from zinc ore, cadmium pollution can be expected in these areas (Goeller, Hise, and Flora, 1973). In addition, large urban and industrial areas often have a higher cadmium content in the environment due to various industrial processes involving products containing cadmium (Sections 7.3.2, 7.3.3, 7.3.4, and 7.3.5). The exact chemical form of cadmium

TABLE 7.3. APPROXIMATE MATERIAL BALANCE FOR CADMIUM IN THE UNITED STATES IN 1968[a]

Source	Cadmium metal		Cadmium in emissions					
			Air		Soil and water		Total	
	(10^3 kg)	(10^3 lb)	(10^3 kg)	(10^3 lb)	(10^3 kg)	(10^3 lb)	(10^3 kg)	(10^3 lb)
Domestic zinc ores	2190	4,827						
Domestic lead and copper ores	216	477						
Imported zinc ores processed in United States	2816	6,208						
Imported lead ores processed in United States	74	163						
Imported flue dusts processed in United States	728	1,605						
Total cadmium metal available	6024	13,280						
Emissions from extraction, refining, and production			953	2100	294	649	1247	2749
Remaining in zinc (probable upper limit of cadmium in end product	139	306						
Total domestic production	4638	10,225						
Net import of metal	634	1,397						
Withdrawn from stockpiles	581	1,280						
Recycled from scrap	193	426						
Emission from scrap recovery in recycling				?				
Total metal entering industry	6045	13,328						
Emissions from industrial conversion			15	33		?	15	33
Total metal entering consumer channels	6030	13,294						
Total emissions from mining, production, and conversion							1262	2782
Total emissions from consumption and disposal			186	410	909	2004	1095	2415
Total metal into "permanent use" and wastes with unknown disposition	4934	10,879						

(continued)

TABLE 7.3 (continued)

Source	Cadmium metal		Cadmium in emissions					
			Air		Soil and water		Total	
	(10^3 kg)	(10^3 lb)	(10^3 kg)	(10^3 lb)	(10^3 kg)	(10^3 lb)	(10^3 kg)	(10^3 lb)
Emissions from cadmium content of fossil fuels			132-1980	290-2200				
Emissions from cadmium content of fertilizer					23-227	50-500		
Emissions from cadmium content of tires			5	11				
Emissions from cadmium content of lube oil			0.8	1.8		?		
Total							160-1231	353-2718
Total emissions to air, soil, and water							2518-2588	5550-7910

Source: Adapted from Goeller, Hise, and Flora, 1973, Table IV-7, p. 87.

TABLE 7.4. CADMIUM RELEASED FROM VARIOUS SOURCES IN THE UNITED STATES

Source	Cadmium released (metric tons)	(short tons)	Percentage of total
Copper mining	Negligible	Negligible	Negligible
Zinc mining	<0.9	<1	0.01
Lead mining	Negligible	Negligible	Negligible
Primary copper			
Roasting	208	229	7.59
Reverberatory furnace	85	94	3.12
Converters	245	270	8.95
Material handling	53	59	1.96
Primary zinc			
Roasting	604	666	22.07
Sintering	258	284	9.41
Distillation	82	90	2.98
Material handling	Negligible	Negligible	Negligible
Primary nickel	Negligible	Negligible	Negligible
Primary lead			
Sintering	60	66	2.19
Blast furnace	17	19	0.63
Reverberatory furnace	2.7	3	0.10
Material handling	Negligible	Negligible	Negligible
Secondary copper			
Sweating furnace	64	70	2.32
Blast furnace	50	55	1.82
Iron and steel, blast furnace	907	1000	33.14
Nonferrous alloys			
Furnaces	2.7	3	0.10
Material handling	Negligible	Negligible	Negligible
Cadmium paint pigments	10	11	0.36
Cadmium-barium plastic stabilizers	2.7	3	0.10
Cadmium-nickel batteries	<0.9	<1	0.01
Miscellaneous cadmium products	<0.9	<1	0.02
Use of pesticides, herbicides, and fungicides	<0.9	<1	0.01
Fertilizer application	<0.9	<1	0.02
Incinerators	86	95	3.15
Total	2738	3018	100.06

Source: Adapted from The MITRE Corporation, as cited in Goldberg, 1973, p. 105.

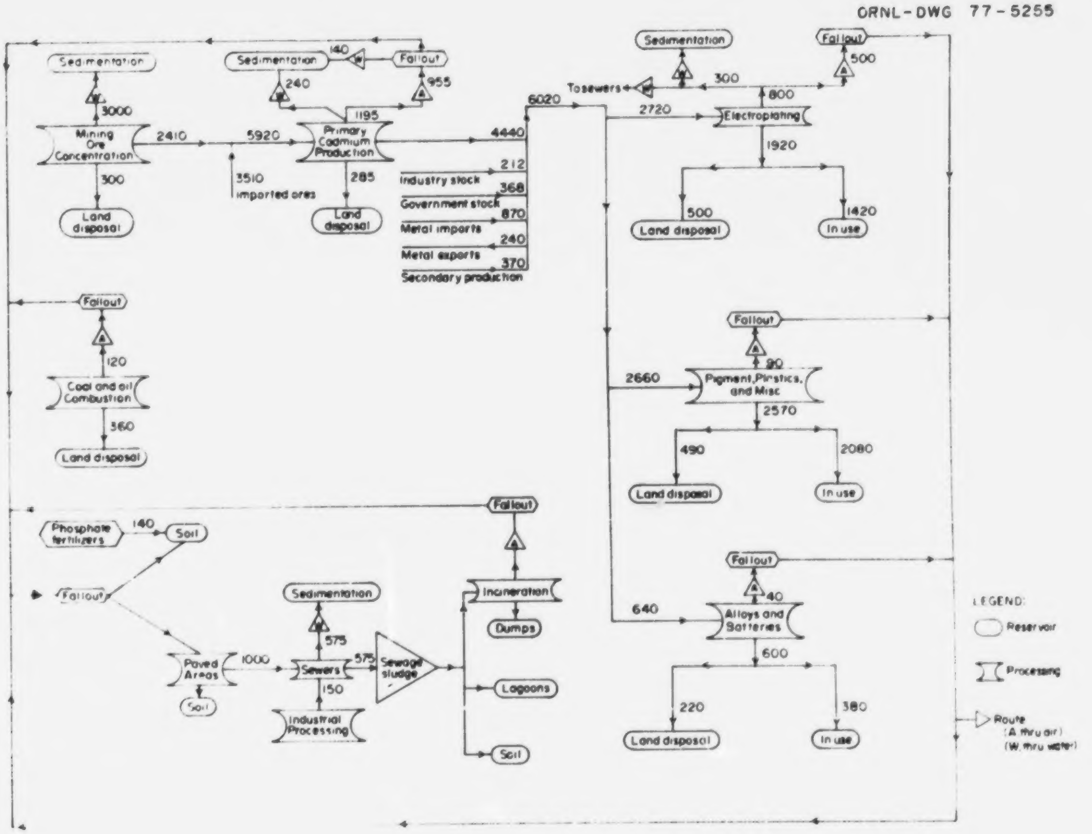

ORNL-DWG 77-5255
Sedimentation
3000
Mining Ore Concentration
2410
300
Land disposal
Sedimentation
140
Fallout
240
955
1195
5920
3510 imported ores
Primary Cadmium Production
285
Land disposal
4440
212
Industry stock
368
Government stock
870
Metal imports
240
Metal exports
370
Secondary production
6020
Sedimentation
Fallout
500
To sewers
300
800
2720
Electroplating
1920
500
Land disposal
1420
In use
Fallout
120
Coal and oil Combustion
360
Land disposal
Fallout
90
2660
Pigment, Plastics, and Misc
2570
490
2080
Land disposal
In use
Fallout
Phosphate fertilizers
140
Soil
Fallout
Paved Areas
1000
Soil
Sedimentation
575
Sewers
575
Sewage sludge
150
Industrial Processing
Incineration
Dumps
Lagoons
Soil
Fallout
40
640
Alloys and Batteries
600
220
380
Land disposal
In use
LEGEND:
Reservoir
Processing
Route (A thru air) (W thru water)

Figure 7.3. Rates, routes, and reservoirs of cadmium in the environment. Values are calculated in tons per year for the United States. Source: Fleischer et al., 1974, Figure 5, p. 282.

released into the environment and the medium to which it is released depend on the particular industry. Natural and elevated concentrations of cadmium found in air, land, and water are considered in the next three sections.

7.3.2 Distribution in Air

Because cadmium is a toxic element, there is considerable interest in air pollution emissions from various industries. Air emissions of cadmium [estimated to be about 1500 metric tons (1680 short tons) for 1968; see, however, somewhat higher values in Table 7.3] occur in smelting of various lead, copper, and zinc ores, in incineration of certain industrial and municipal wastes, in combustion of coal and oil, and through electroplating, pigments, and plastic industries (Fleischer et al., 1974). Questions of concern include the form and concentration of cadmium in the air and the distribution in rural and urban areas. Presently, little information exists on the exact chemical form of cadmium in air. Some cadmium is probably released as dust, most likely as CdS or $CdSO_4$, during mining and conversion

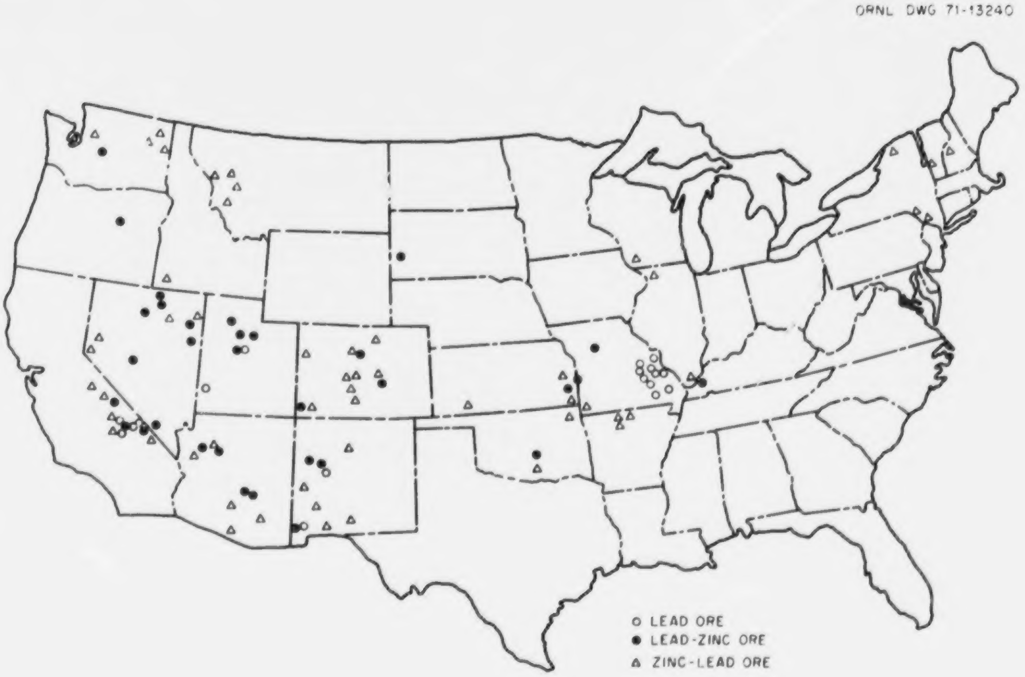

ORNL DWG 71-13240
○ LEAD ORE
● LEAD-ZINC ORE
△ ZINC-LEAD ORE

Figure 7.4. Location of U.S. deposits of lead, lead-zinc, and zinc-lead ore. Source: Goeller, Hise, and Flora, Figure IV-5, p. 117.

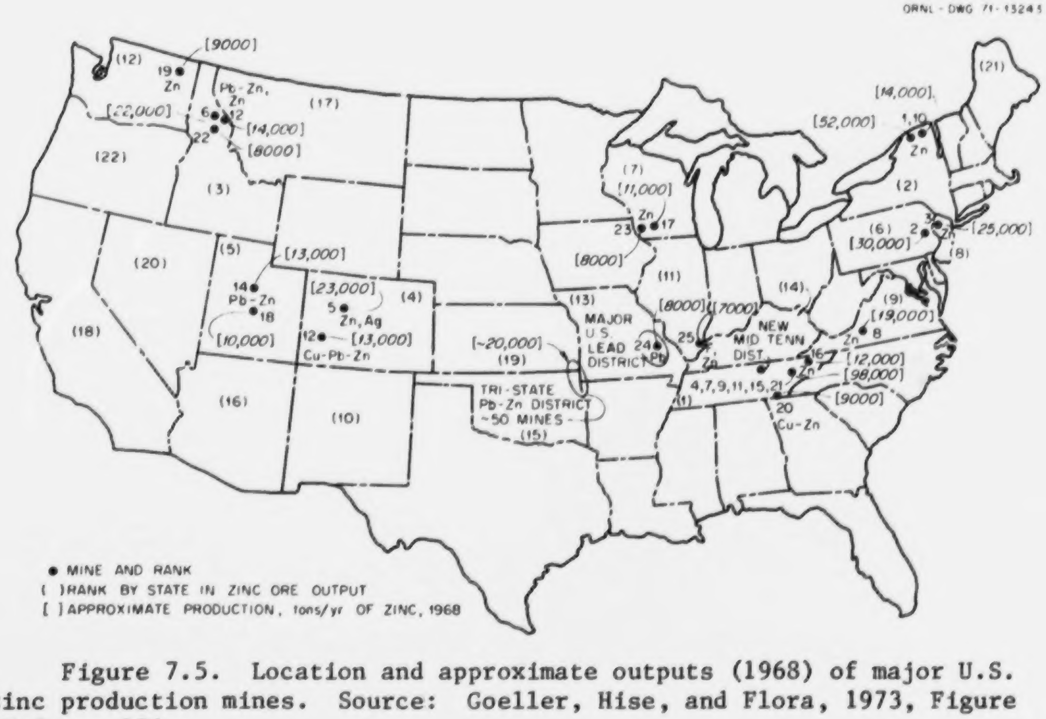

ORNL - DWG 71-13243
(12)
[9000]
19
Zn
Pb-Zn,
Zn
(17)
[22,000]
6
12
22
[14,000]
[8000]
(22)
(3)
(20)
(18)
(5)
[13,000]
14
Pb-Zn
18
[10,000]
[23,000]
5
Zn, Ag
(4)
12
[13,000]
Cu-Pb-Zn
(16)
(10)
[~20,000]
(19)
TRI-STATE
Pb-Zn DISTRICT
~50 MINES
(15)
(13)
MAJOR
U.S.
LEAD
DISTRICT
24
Pb
(7)
[11,000]
Zn
23
17
[8000]
(11)
[8000]
[7000]
25
Zn
(1)
NEW
MID TENN
DIST.
(14)
4,7,9,11,15,21
16
Zn
20
Cu-Zn
[12,000]
[98,000]
[9000]
(9)
[19,000]
Zn
8
[52,000]
[14,000]
1,10
Zn
(21)
(2)
(6)
2
3
Zn
[30,000]
[25,000]
(8)
● MINE AND RANK
( ) RANK BY STATE IN ZINC ORE OUTPUT
[ ] APPROXIMATE PRODUCTION, tons/yr OF ZINC, 1968

Figure 7.5. Location and approximate outputs (1968) of major U.S. zinc production mines. Source: Goeller, Hise, and Flora, 1973, Figure IV-6, p. 124.

processes, and some is probably released as metal vapor during the smelting process. Such cadmium probably reacts with oxygen to form cadmium oxide (Section 2.3.1).

The concentration of cadmium in air around point sources can be considerable. Friberg et al. (1974) cited data showing a weekly mean of 0.3 μg/m³ at 500 m from a Swedish factory using copper-cadmium alloys and a maximum 24-hr average of 3 μg/m³ near a Japanese smelter. At 500 m from another Japanese smelter, average values lay between 0.16 and 0.32 μg/m³. The most common example used for the United States is East Helena, Montana, which had three-month averages of 0.06 and 0.29 μg/m³ at 1300 and 800 m from the smelter. Such concentrations usually decrease with distance from the source due to dispersion and dust fall or precipitation (Friberg et al., 1974).

Although elevated cadmium levels are often found in the vicinity of zinc smelters, the levels may not be high near lead smelters. De Koning (1974) found that cadmium levels in air, soil, and plants near a lead smelter were slightly higher than normal, although not arranged geographically to suggest that the smelter was a point source of cadmium pollution. In contrast, Lagerwerff, Brower, and Biersdorf (1973) suggested that elevated cadmium levels, especially around smelters which also process zinc ore, indicate that the smelter is a point source. In addition, high concentrations of cadmium in soils and litter were found near the American Metals Climax, Inc. lead smelter in southeastern Missouri; these concentrations decreased with increasing distance from the smelter (Andren et al., 1974).

There appears to be little argument that the elevated concentrations in urban areas are due to industrial activity (Fleischer et al., 1974). No data were found on the contribution of various natural processes to the cadmium content in air. The annual range for airborne particulate cadmium in New York City was 0.006 to 0.002 μg/m³ in the Bronx, 0.009 to 0.036 μg/m³ in lower Manhattan, and 0.001 to 0.005 μg/m³ in Tuxedo, New York (detection limit of 0.001 μg/m³) (Kneip et al., 1970). In lower Manhattan the concentration showed a seasonal dependence: November through February had lower concentrations, perhaps because of higher average wind speed during these months. In a study of airborne particulates in the Chicago—northwest Indiana region (50 sampling stations, six times during the summer), the suburban areas yielded about 0.006 μg/m³ compared to values for the Lake Michigan shoreline industrial areas of 0.015 to 0.080 μg/m³ (Harrison and Winchester, 1971). These authors suggested that the cadmium in this region comes from local sources and that, except for one station, it probably arises from gasoline and coal emissions. This conclusion is based on the fact that combustion of coke, fuel oil, and gasoline produces emissions with ratios of cadmium to lead, cadmium to copper, and copper to lead similar to those found in the air samples. The low concentrations in suburban areas indicate that the emissions are dissipated rather rapidly. A study of trace-element air pollution in 77 midwestern cities by dust fall analysis showed that industrial areas had the highest amount of cadmium fallout (0.075 mg/m²-month) compared to commercial and residential areas, which had 0.063 and 0.040 mg/m²-month respectively (Hunt et al., 1971). Tables 7.5 and 7.6 present data from the National Air Surveillance Network for the cadmium content of urban and

TABLE 7.5. CADMIUM CONCENTRATIONS IN U.S. URBAN AIR, 1969 ($\mu g/m^3$)

Location	Yearly average	Range
Alabama		
Gadsden		0-0.003
Huntsville	0.003	0-0.003
Mobile	0.007	0.004-0.010
Montgomery	0.004	0-0.005
Alaska		
Anchorage	0.003	0-0.004
Fairbanks	0.004	0.003-0.004
Arizona		
Maricopa County	0.003	0-0.004
Phoenix	0.006	0.005-0.009
Tucson	0.008	0.004-0.012
Arkansas		
Little Rock		0-0.006
Texarkana		0-0.004
West Memphis	0.004	0-0.008
California		
Anaheim	0.003	0-0.005
Burbank	0.004	0.003-0.005
Fresno	0.003	0-0.005
Glendale		0-0.004
Long Beach	0.006	0.004-0.011
Los Angeles	0.006	0.006
Oakland	0.007	0.005-0.011
Ontario	0.007	0.004-0.010
Riverside	0.005	0.004-0.005
Sacramento	0.004	0.004-0.005
San Bernardino	0.013	0.006-0.020
San Diego	0.010	0.006-0.015
San Francisco		0-0.006
San Jose	0.004	0.003-0.005
Santa Ana		0-0.006
Torrance	0.005	0-0.007
Colorado		
Denver	0.018	0.015-0.027
Montezuma County		0

(continued)

TABLE 7.5 (continued)

Location	Yearly average	Range
Connecticut		
Bridgeport	0.012	0.008-0.018
Hartford	0.006	0.005-0.006
New Haven	0.007	0.005-0.009
Waterbury	0.020	0.008-0.029
Delaware		
Wilmington	0.014	0.006-0.025
District of Columbia	0.008	0.006-0.010
Florida		
Jacksonville	0.005	0.003-0.006
Miami	0.008	0.005-0.011
St. Petersburg	0.003	0.003-0.004
Tampa	0.006	0.005-0.007
Georgia		
Atlanta	0.007	0.006-0.011
Columbus		0-0.004
Savannah	0.004	0.003-0.004
Hawaii		
Honolulu		0
Idaho		
Boise City		0-0.008
Illinois		
Chicago	0.015	0.014-0.015
East St. Louis	0.022	0.013-0.045
Joliet	0.009	0.005-0.012
North Chicago	0.007	0.004-0.011
Peoria	0.011	0.008-0.015
Rockford	0.013	0.010-0.016
Springfield	0.005	0.004-0.007
Indiana		
East Chicago	0.028	0.017-0.046
Evansville	0.008	0.007-0.009
Fort Wayne	0.008	0.007-0.015
Gary	0.009	0.008-0.012
Hammond	0.011	0.010-0.012
Indianapolis	0.015	0.005-0.025
New Albany	0.005	0.004-0.006
South Bend	0.006	0.006-0.007
Terre Haute	0.005	0.005-0.007

(continued)

TABLE 7.5 (continued)

Location	Yearly average	Range
Iowa		
Davenport	0.010	0.007-0.016
Des Moines	0.005	0.004-0.005
Dubuque	0.009	0.004-0.013
Kansas		
Kansas City	0.010	0.007-0.012
Topeka	0.005	0.003-0.006
Wichita	0.005	0.004-0.006
Kentucky		
Ashland	0.023	0.017-0.026
Covington	0.011	0.007-0.013
Louisville	0.006	0.006
Louisiana		
Baton Rouge	0.003	0.003
New Orleans	0.004	0.004-0.005
Shreveport	0.004	0.003-0.004
Maryland		
Baltimore	0.011	0.008-0.017
Massachusetts		
Boston	0.006	0.004-0.008
Fall River	0.005	0.003-0.007
Springfield	0.006	0.004-0.009
Worchester	0.006	0.003-0.008
Michigan		
Dearborn	0.011	0.007-0.013
Detroit	0.012	0.010-0.015
Flint	0.007	0.006-0.009
Grand Rapids	0.012	0.009-0.018
Lansing	0.004	0.003-0.005
Saginaw	0.003	0.003-0.005
Trenton	0.006	0.004-0.007
Minnesota		
Duluth		0-0.004
Minneapolis	0.007	0.006-0.008
St. Paul	0.009	0.006-0.015
Missouri		
Kansas City	0.007	0.004-0.010
St. Louis	0.036	0.013-0.066
Montana		
Helena	0.028	0.005-0.077

(continued)

TABLE 7.5 (continued)

Location	Yearly average	Range
Nebraska		
Omaha	0.004	0.003-0.005
Nevada		
Las Vegas		0-0.004
Reno		0-0.005
New Hampshire		
Concord		0
New Jersey		
Burlington County	0.008	0.006-0.009
Elizabeth	0.017	0.009-0.029
Glassboro	0.010	0.009-0.013
Hamilton	0.007	0.005-0.009
Jersey City	0.012	0.010-0.015
Newark	0.038	0.014-0.097
Paterson	0.006	0.004-0.007
Perth Amboy	0.018	0.011-0.024
Trenton	0.010	0.008-0.012
New Mexico		
Albuquerque	0.004	0-0.007
New York		
Albany	0.003	0-0.004
Buffalo	0.007	0.006-0.008
New York City	0.014	0.004-0.023
Niagara Falls	0.006	0.004-0.007
Rochester	0.008	0.004-0.011
Syracuse	0.008	0.003-0.010
Utica	0.003	0-0.005
North Carolina		
Charlotte	0.004	0-0.005
Durham		0-0.003
Greensboro	0.006	0.005-0.008
Winston-Salem	0.005	0.004
North Dakota		
Bismarck		0
Ohio		
Akron	0.008	0.007-0.010
Canton	0.008	0.006-0.009
Cincinnati	0.016	0.013-0.019
Cleveland	0.015	0.008-0.024
Columbus	0.013	0.008-0.021
Dayton	0.010	0.008-0.011

(continued)

TABLE 7.5 (continued)

Location	Yearly average	Range
Toledo	0.005	0.005
Youngstown	0.012	0.009-0.015
Oklahoma		
Oklahoma City		0-0.005
Tulsa	0.013	0.008-0.025
Oregon		
Medford		0-0.004
Portland	0.004	0.003-0.004
Pennsylvania		
Allentown	0.020	0.011-0.028
Bethlehem	0.023	0.015-0.029
Erie	0.009	0.007-0.012
Harrisburg	0.005	0.004-0.006
Hazelton	0.005	0.004-0.008
Johnstown	0.008	0.005-0.010
Philadelphia	0.015	0.010-0.020
Pittsburgh	0.014	0.009-0.020
Reading	0.011	0.008-0.014
Scranton	0.008	0.007-0.009
Warminster	0.006	0.005-0.008
West Chester	0.008	0.004-0.010
Wilkes-Barre	0.008	0.006-0.012
York	0.006	0.004-0.008
Puerto Rico		
Bayamon	0.006	0.006-0.008
Catano	0.003	0-0.004
Guayanilla		0
Ponce		0
San Juan		0-0.003
Rhode Island		
East Providence	0.008	0.007-0.010
Providence	0.005	0.004-0.005
South Carolina		
Columbia		0-0.003
Greenville	0.007	0.007-0.008
Tennessee		
Chattanooga	0.011	0.010-0.014
Knoxville	0.004	0-0.005
Memphis	0.004	0.003-0.005
Nashville	0.004	0.003-0.004

(continued)

TABLE 7.5 (continued)

Location	Yearly average	Range
Texas		
Dallas	0.005	0.003-0.008
El Paso	0.105	0.057-0.150
Fort Worth	0.004	0-0.006
Houston	0.004	0-0.005
Pasadena		0-0.006
San Antonio	0.003	0-0.003
Utah		
Ogden	0.007	0.005-0.012
Salt Lake City	0.009	0.003-0.016
Vermont		
Burlington	0.005	0.004-0.006
Virginia		
Danville	0.003	0-0.004
Hampton	0.003	0-0.005
Lynchburg	0.012	0.008-0.021
Newport News	0.007	0.004-0.009
Norfolk	0.004	0.003-0.005
Portsmouth	0.008	0.007-0.009
Richmond	0.004	0.004-0.005
Roanoke	0.012	0.009-0.014
Washington		
Seattle	0.008	0.003-0.019
Spokane	0.004	0-0.007
Tacoma	0.010	0.005-0.016
West Virginia		
Charleston	0.006	0.005-0.007
Wisconsin		
Eau Claire		0-0.004
Kenosha	0.008	0.004-0.010
Madison	0.004	0.003-0.004
Milwaukee	0.010	0.006-0.017
Racine	0.006	0.005-0.007
Superior	0.003	0-0.004
Wyoming		
Casper		0
Cheyenne	0.005	0.004-0.006

Source: Adapted from U.S. Environmental Protection Agency, 1973, Table 4-3, pp. 4-9 — 4-12.

TABLE 7.6. NONURBAN SITES SHOWING LESS THAN 0.001 µg CADMIUM PER CUBIC METER OF AIR IN 1969

- Arizona
  - Grand Canyon National Park
- Arkansas
  - Montgomery County
- California
  - Humboldt County
- Florida
  - Hardee County
- Idaho
  - Butte County
- Indiana
  - Monroe County
  - Parke County
- Maine
  - Acadia National Park
- Maryland
  - Calvert County
- Missouri
  - Shannon County
- Montana
  - Glacier National Park
- Nebraska
  - Thomas County
- Nevada
  - White Pine County
- New Hampshire
  - Coos County
- New York
  - Jefferson County
- North Carolina
  - Cape Hatteras National Park
- Oklahoma
  - Cherokee County
- Oregon
  - Curry County
- Pennsylvania
  - Clarion County
- Rhode Island
  - Washington County
- South Carolina
  - Richland County
- South Dakota
  - Black Hills National Forest
- Tennessee
  - Cumberland County
- Texas
  - Matagorda County
- Vermont
  - Orange County
- Virginia
  - Shenandoah National Park
  - Wythe County
- Wisconsin
  - Door County
- Wyoming
  - Yellowstone National Park

Source: Adapted from U.S. Environmental Protection Agency, 1973, Table 4-4, p. 4-13.

nonurban areas in 1969 (U.S. Environmental Protection Agency, 1973). The significant concentrations of cadmium observed in cities and, more particularly, in industrial areas suggest that the source of cadmium in air is anthropogenic.

Forms and particle-size distribution of cadmium in air are important in assessing hazards from emission sources. Samples of air (taken on different days) from Cincinnati, Ohio, and suburban Fairfax (9 miles away) showed that cadmium was four times higher in the city (0.08 $\mu g/m^3$) than in the suburb (0.02 $\mu g/m^3$). The mass median diameter was larger for particles in the suburb (estimated 10 μm) than in the city (3.1 μm). Since about 40% of the particles in the city had sizes less than 2 μm, significant absorption through the lung would be expected (Lee, Patterson, and Wagman, 1968).

Particle size and cadmium concentration were examined in fly ash from the precipitating system and from stack emissions of eight power plants in the United States. The findings indicate that the highest concentrations of cadmium occur in the smallest airborne particles (e.g., 35 ppm in ash from 1.1- to 2.1-μm particles, 22 ppm in ash from 3.3- to 4.7-μm particles, and 15 ppm in ash from 7.3- to 11.3-μm particles). In precipitated fly ash the cadmium concentrations were less than 10 ppm for all particle sizes, suggesting that (1) existing particle collection devices allow the emission of small toxic particles and (2) estimates of emission concentrations determined from precipitated fly ash may "grossly underestimate the actual emissions" (Natusch, Wallace, and Evans, 1974). Why the highest concentration of cadmium is associated with the smallest particles is not precisely known. A possible explanation may be that cadmium is volatilized at higher temperatures and is then adsorbed or condensed onto the available surface area of airborne particles (Natusch, Wallace, and Evans, 1974). Smaller particles with greater surface-to-mass ratios therefore adsorb more cadmium than larger particles.

Information on the concentration of different-sized particles in the air is also needed to assess the potential hazard of an emission source. Toca, Cheever, and Berry (1973) showed an increasing cadmium concentration with decreasing particle size in the effluent from a coal-fired boiler and, in addition, found that the last stages of their sampling train, containing the smallest trapped particles, yielded the greatest weight per cubic meter of sample volume.

7.3.3 Distribution in Soils

The distribution of cadmium in soils is a more complex problem than distribution in air because parent rock may contain cadmium compounds, cadmium may be surface deposited by settling from air, and cadmium may become more or less tightly bound to soil particles. The soil also is a more permanent reservoir for cadmium, and concentrations may be expected to increase with time.

The cadmium content in unpolluted soil is related to the concentration of cadmium in natural parent materials of the soil. Tables 7.7 and 7.8 illustrate the considerable range in cadmium concentrations in igneous and sedimentary rocks. Vinogradov (cited in Baes, 1973) reported that zinc and cadmium contents of soils vary with the nature of the parent rock, being high in soils on basaltic rock and low in soils on granitic rock. In addition to natural variations, human activity greatly influences cadmium content of soils (Table 7.9); for example, uncontaminated soils have average values of 0.4 ppm or less compared with an average of 72 ppm in soil near a smelter (Fleischer et al., 1974). As another example, examination of 5-cm

TABLE 7.7. CADMIUM IN SEDIMENTARY ROCKS

Sedimentary rock	Number of samples	Cadmium content (ppm)		
		Range	Average	Median
Limestones	23		0.10	
Sandstones (composite of 14)			<0.03	
Shales	103	<0.3-11.0	1.4[a]	0.8
Organic content <0.5%		<0.3-0.8	0.35	0.3
Organic content 0.5%-1.0%		0.3-1.8	0.8	0.7-0.8
Organic content >1%		0.5-8.4	2.0	1.2
Oceanic sediments	189	0.04-1.88	0.5[b]	
Lake sediments	26	5-19	11.	
Oceanic manganese oxides	34	<3-21.2	8	5.3
Phosphorites	163	<10-500	25	

[a] Marowsky and Wedepohl (1971) gave 0.3 ppm.

[b] Aston et al. (1972) reported 0.04-0.58 ppm (av 0.226 ppm) in 35 Atlantic deep-sea sediments.

Source: Adapted from Fleischer et al., 1974, Table 3, p. 260.

TABLE 7.8. CADMIUM IN IGNEOUS ROCKS

Igneous rocks	Number of samples analyzed	Cadmium content (ppm)		Zn/Cd ratio
		Range	Average	
Rhyolitic	43	0.03-0.57	0.23	480-177
Granitic	43	0.01-1.6	0.2	90-7000
Intermediate	4	0.017-0.32		
Basalt, diabase, gabbro	79	0.01-1.00	0.13	80-3600
Peridotite	3	<0.001-0.029	0.03	
Dunite	1	0.005-0.154		
Eclogite	4	0.03-1.6	0.1	
Alkalic rock	7	0.04-0.90	0.25	27-1430

Source: Adapted from Fleischer et al., 1974, Table 2, p. 260.

TABLE 7.9. CADMIUM IN SOILS

Locality and type of soil	Number of samples	Depth (cm)	Cadmium content (ppm)	
			Range	Average
		Normal soils		
U.S.S.R., tundra, podsols, forest, red earth	40	A, B, and C horizons	0.01-0.07	0.06
Poland, distant from industrial areas	33	0-20	0.04 (max)	0.016
Wales, Ystwyth Valley				1.0
Maryland, Missouri, Ohio, 32 m from highways[a]	4	10-15	0.12-0.52	0.26
Helena Valley, Montana, 18-60 km from smelter	17	6-10	<0.5-2[b]	1.4[b]
Annaka City Japan, 900 m from refinery	2	40-60	0.3-0.4	0.35
Southwest Wales	4		0.3-0.5	0.4
Lower Fraser Valley, British Columbia	33	Surface		0.88
		Contaminated soils		
Poland, industrial areas	67	0-20	0.3-0.8 (max)	0.17-0.28
Wales, Ystwyth Valley, contaminated			1.5-3.0	
Maryland, Missouri, Ohio, 8 m from highways[a]	4	0.5	0.90-1.82	1.28
Helena Valley, Montana, 1 km from smelter	7	0.10	26-160	72
Annaka City, Japan				
150-250 m from smelter		Surface	23-88	
900 m from smelter		5	44	
Poland, 600 m from zinc metallurgical factory	1 1	0-10 15-30	250 110	
Southwest Wales, Swansea, 1.5 km from contaminated area			26	
Bartlesville, Oklahoma, 1500 ft from smelter		12.5-27.5	450	
British Columbia, 15 m from smelter	4	Surface	7.9-95.2	49.0
Grand Rapids area, Michigan				
Residential area	70	0-5		0.41
Agricultural area	91	0-5		0.57
Industrial area	86	0-5		0.66
Airport	7	0-5		0.77

[a] Samples collected near U.S. 1, Beltsville, Maryland; Washington-Baltimore Parkway, Bladensburg, Maryland; Interstate 29, Platte City, Missouri; Seymour Road, Cincinnati, Ohio.

[b] These values probably reflect some contamination.

Source: Adapted from Fleischer et al., 1974, Table 6, p. 264. Data collected from several sources.

cores of surface soil about Grand Rapids, Michigan, gave average values of 0.77 ppm for airport locales, 0.66 ppm for industrial locales, 0.57 ppm for agricultural soils, and 0.41 ppm for residential areas. The same general trend was found for nine other trace elements (Klein, 1972).

Accumulations of cadmium in the vicinity of lead and zinc smelters have been reported. Samples from near a lead smelter in Galena, Kansas, showed that surface soil contamination decreased from 102 ppm at 330 m from the smelter to 32.6 ppm at 1000 m and 26.8 ppm at 1670 m. At each locale the cadmium concentration decreased with increasing soil depth. At the 330-m site, cadmium concentrations were 102 ppm at 0 to 5 cm, 48 ppm at 5 to 10 cm, and 22 ppm at 10 to 22 cm; the corresponding values at 1000 m were 32.6, 12.2, and 3.1 ppm respectively (Lagerwerff, Brower, and Biersdorf, 1973). A similar trend was observed in Annaka City, Japan, site of a large zinc refinery; surface soil samples contained 31 ppm cadmium (Kobayashi, 1972). Surface garden soils in Tacoma, Washington, contained 7 to 12 ppm cadmium near a copper smelter and about 1 to 2 ppm at distances of 1.6 to 3.2 km; "normal" soils were reported to contain 0.06 ppm cadmium (Ratsch, 1974).

Concentrations of cadmium in the litter horizons of soil near the American Metals Climax, Inc. lead smelter decreased from about 100 ppm 0.4 km from the smelter to about 20 ppm at 2 km (Andren et al., 1974). The average nitric acid—soluble cadmium content of 33 agricultural soils in the Lower Fraser Valley, British Columbia, Canada, was 0.88 ppm; however, in the vicinity of a battery smelter the surface cadmium concentration was as high as 95 ppm. Vertical distribution in the soil dropped rapidly from 44 ppm at the surface (15 m from the smelter) to 2.45 ppm at the 5- to 10-cm depth and to 0.67 ppm at the 10- to 15-cm depth (John, Chuah, and VanLaerhoven, 1972).

In addition to cadmium contamination of soil by fallout of particulate cadmium from smelters and from the combustion of coal and oil, portions can be added during fertilization with superphosphate or any fertilizer containing phosphate. Cadmium is present in phosphate rock and is concentrated during production of phosphate fertilizer (acidulation or electric furnace processes) (Goeller, Hise, and Flora, 1973). Because plants take up cadmium, the presence of "excess" cadmium in the soil will lead to higher concentrations in plants (Section 4.2.1). Increased plant uptake of cadmium can be a potential hazard if it results in increased human uptake. Examination of cadmium content in several Swedish fertilizers gave values of between 6 and 30 mg/kg fertilizer. With the recommended fertilizer application rate of up to 600 kg/ha, 4 to 18 g cadmium may be added per hectare. This is comparable to the amount of cadmium which would be added through the application of the recommended amount of sewage sludge as fertilizer (Stenström and Vahter, 1974). Williams and David (1973) reported the cadmium content of New South Wales, Australia, fertilizers to lie between 18 and 91 ppm and to be correlated with the phosphorus content of the fertilizer. The cadmium was available for plant uptake and tended to accumulate in an exchangeable form in the upper 10 cm of soil.

Sewage sludge can be used for soil improvement programs, but one major concern is the fate of heavy metals within the sludge. The Swedish Board of

Health and Welfare recommended that sludge with greater than 15 mg cadmium per kilogram (dry weight) not be used for soil improvement and that the application rate be less than 1 ton/ha (corresponding to about 15 g cadmium per hectare). Stenström and Lösjö (1974), in a report of a long-term experiment, suggested that cadmium uptake into wheat grain is proportional to the total amount of cadmium in the soil. They speculated that if availability does not decrease with time and if yearly sludge applications are used, cadmium concentrations in foods "within a few decades" will be sufficiently high to cause daily cadmium uptake by humans to reach the limit suggested by the World Health Organization (60 to 70 μg/day). On the other hand, there is evidence to suggest that availability of sludge-borne cadmium for assimilation by plants may decrease with time after application (Lue-Hing et al., 1977) (Sections 4.2.2.1.1 and 7.5).

Studies of metals in soils and plants along roadsides have been used to determine whether motor vehicles are a source of lead, zinc, nickel, and cadmium pollution. Lagerwerff and Specht (1970) concluded that contamination might be related to traffic because the soil concentration of each metal decreased with distance from the road. Surface soil concentrations of cadmium were 1.45, 0.40, and 0.22 mg/kg dry weight at distances of 8, 16, and 32 m from the road. Similar soil concentrations were found in three other roadside plots. Conversely, in another study Creason et al. (1972) concluded from analyses of cadmium in dust fall that no concentration gradient exists near roads and, therefore, that automobiles may not be a major or specific source of cadmium pollution.

7.3.4 Distribution in Water

As in air and soil, the concentration of cadmium in water varies with season and with distance from the input source. Tables 7.10 and 7.11 (Fleischer et al., 1974) list concentrations of cadmium in seawater and fresh water. Since several factors can influence concentrations in particular locales, different types of water are individually discussed.

There are several reports on the heavy-metal content in water from mining and smelting areas. The cadmium content of the Biala and Sztola streams in Poland ranged from 0.8 to 16.7 ppb, with the highest values below the mouth of industrialized channels of the region. The highest values for the streams occurred in the autumn-winter period. The mine waters contained from 1.3 to 3.7 ppb cadmium (Pasternak, 1973). The concentration of cadmium in water in the lead-zinc mining and industrial area of Springfield, Missouri, was from 0.11 to 1.22 ppb in surface water, from 0 to 1.66 ppb in shallow wells, and from 0.35 to 1.06 ppb in deep wells. In the Joplin, Missouri, area, mean cadmium concentrations were 1.6 ± 3.8 ppb in surface water, 1.7 ± 2.4 ppb in shallow well water, and 0.5 ± 0.7 ppb in deep well water (Proctor et al., 1974). The Canyon Creek Basin mining area in Idaho had concentrations of cadmium as high as 20 ppb in tailing pond effluents and also had elevated levels in groundwater (200 to 400 ppb) and surface water (40 ppb) due to leaching from old tailings buried in the basin area (Mink, Williams, and Wallace, 1972).

TABLE 7.10. CADMIUM IN WATERS

Locality	Analytical method	Number of samples		Cadmium content (µg/liter, approx ppb)		
		Analyzed	Cadmium not found[a]	Range	Average	Median
	Seawater					
Caribbean Sea		1	0		0.3	
Pacific Ocean off California	Spectrographic after concentration	24	24	<0.7		
Irish Sea	Dithizone	5	0	0.032-0.075	0.055	
Surface	Dithizone	12	0	0.045-0.232	0.166	
Irish Sea and English Channel	Polarographic			0.6-3.0	1.3	
English Channel	Atomic absorption	3	0	0.081-0.180	0.119	
	Atomic absorption after concentration	4		<0.01-0.38		0.06
Irish Sea						
Western part	Atomic absorption after concentration	10		<0.01-0.52		0.11
Eastern part	Atomic absorption after concentration	21		0.01-0.62		0.04
Atlantic, near Scotland	Atomic absorption after concentration	8		<0.01-0.18		<0.01
Atlantic, NW Ireland	Atomic absorption after concentration	5		<0.01-0.41		0.04
North Sea	Stripping voltammetry	4		0.29-0.60		0.41
Atlantic (?)	Atomic absorption				0.18	
Menai Straits	Atomic absorption after concentration	5	1		0.06	
Atlantic, off coast of Africa	Atomic absorption after concentration	101		0.07-0.71	0.11	
Gulf of Finland	Atomic absorption after concentration	6		0.1-2.2	0.7	0.4-0.5
Gulf of Bothnia	Stripping anodic voltammetry	6		0.3-10.2	2.9	0.5-0.6
Oslo fjord, depth 40 m	Stripping anodic voltammetry	5		0.12-0.29	0.19	
Coast off Wakayama Prefecture, Japan	Colorimetric after concentration			0.08-0.17		
Seto Inland Sea, Japan	Atomic absorption after concentration			0.1-0.8		
	Fresh water					
Surface waters, United States	Atomic absorption spectra and spectrographic	569	2165	<1-130		<1
Surface waters, Siberia	Spectrographic after concentration	2490	3474	<1-207	0.032	
Lake Erie	Atomic absorption spectra	14	12	0-1		
Lake Ontario	Atomic absorption after concentration	9	0	0.03-0.15	0.09	
Linsley Pond, Connecticut	X-ray fluorescence	6	0	0.71-2.08	1.15	
"Clean" streams, Wales	Atomic absorption after concentration				0.41	
Well waters, California	Spectrographic after concentration	167	162	<0.7-71		
Hot springs, Arima, Japan	Spectrographic after concentration			0.1-1.0		

[a] The sensitivity is indicated by the lower limit of the range.

Source: Adapted from Fleischer et al., 1974, Table 7, pp. 266-267. Data collected from several sources.

TABLE 7.11. CADMIUM CONCENTRATIONS AND FLOWS IN SELECTED RIVERS

Location	Cadmium content (µg/liter)	Cadmium flow (metric tons per year)	(tons per year)	Zn/Cd ratio	Date
Mississippi-Missouri River[a]					
North Dakota	1	23	25	20	10/9/70
Nebraska	2	76	84	10	10/9/70
	4	152	168	5	10/9/70
Missouri	14	603	665		10/14/70
	8	350	386	0.6	10/14/70
	25	1216	1340	1.6	10/5/70
	8	998	1100		10/13/70
Illinois	16	3946	4350		10/13/70
	12	3030	3340		10/14/70
Missouri	20	5162	5690	1.5	10/15/70
Tennessee	8	1869	2060	16	10/14/70
Louisiana	6	1588	1750		10/20/70
	6	1588	1750		10/20/70
Tributaries to Mississippi-Missouri River					
Ohio River, Kentucky	20	809	892	9.5	10/23/70
Tennessee River, Alabama	90	2794	3080		10/21/70
Illinois River, Illinois	1	18	20	30	10/6/70
Columbia River, Washington	2	178	196	5	10/6/70
Susquehanna River, Pennsylvania	5	46	51	4	10/14/70
Allegheny River, Pennsylvania	1	19	21	60	10/12/70
Sacramento River, California	2	24	27		10/7/70
Mineral Creek, Arizona	30	0.9	1	320	10/8/70

Source: Adapted from Fleischer et al., 1974, Table 24, p. 280.

A regional summary of cadmium in surface water (Table 7.12) shows New England and the Northeast with the highest frequency of detectable levels of cadmium; the highest concentration was found in the Southwest (Durum and Hem, 1972). There is ample documentation of elevated cadmium levels in various surface waters near industrial areas. For example, cadmium concentrations in the Bristol Channel (industrialized area) ranged from 0.28 to 4.20 ppb (mean of 1.13 ppb) (Abdullah, Royle, and Morris, 1972), and in the neighboring Severn Estuary the cadmium concentration ranged from 5.8 ppb near the mouth of the channel to about 0.1 ppb in the adjacent open sea (Butterworth, Lester, and Nickless, 1972). The decrease in cadmium concentration over a relatively short distance suggests that cadmium is being deposited in sediments. Fleischer et al. (1974) concluded that "the surface waters that contain more than a few ppb cadmium near urban areas have almost certainly been contaminated by industrial wastes from metallurgical plants, plating works, or plants manufacturing cadmium pigments, cadmium-stabilized plastics, or nickel-cadmium batteries, or by effluent from sewage treatment."

TABLE 7.12. REGIONAL SUMMARY OF CADMIUM IN U.S. SURFACE WATERS

Region	Cadmium content (ppb)			Cadmium not detected (%)	Cadmium detected (%)
	Max	Min	Median		
New England and Northeast	32	<1	2	36	64
Southeast	90	<1	<1	55	45
Central	40	<1	<1	55	45
Southwest	130	<1	<1	65	35
Northwest	21	<1	<1	78	22

Source: Adapted from Durum and Hem, 1972, Table 7, p. 30. Reprinted by permission of the publisher.

Since there is concern over the heavy-metal content of natural waters, studies have been carried out to estimate concentrations in surface waters that serve as drinking water sources in and near urban and industrial areas. Table 7.13 cites locations in which a U.S. Geological Survey study found that the Public Health Service mandatory upper limit for cadmium concentration (10 ppb) was exceeded; the report concluded that there is no widespread occurrence of cadmium in levels greater than the standard since a large number (720) of collection sites were examined in both industrial and urban areas (Anonymous, 1971). The Community Water Supply Survey of 1969 found that 3 of 969 water supply systems produced finished drinking water with concentrations of cadmium greater than the Public Health Service mandatory limit (10 ppb) (McCabe et al., 1970).

Doolan and Smythe (1973) studied the cadmium content of a variety of waters in New South Wales, Australia. Electrolytic plating wastewater contained cadmium levels as high as 28,500 ppb; very high levels were also found in seepage water from mines. Downriver from these sources, the cadmium content decreased significantly. Tap water contained 0.02 ppb cadmium, but water from unused galvanized piping showed higher levels (1.2 ppb). Galvanized piping (zinc-plated) has been considered as a source for cadmium in drinking water; rates of dissolution depend on the oxygen concentration, pH, and carbonate concentration (Goeller and Flora, 1973) (Section 2). Schroeder et al. (1967) presented data (Table 6.8) showing that tap water contains a higher concentration of cadmium than the water at the inlet, presumably due to dissolution during passage through galvanized piping (Section 2.3.2.4).

There is a lack of data on the chemical nature of cadmium in waters and on whether it is present in soluble or particulate form. Joe Mill Creek in Tennessee flows over carbonate outcroppings which contain sphalerite (ZnS);

TABLE 7.13. LOCATIONS AT WHICH U.S. PUBLIC HEALTH SERVICE MANDATORY UPPER LIMIT FOR CADMIUM IN DRINKING WATER WAS EXCEEDED[a]

State	Location	Metal concentration (ppb)			
		As	Cd	Pb	Hg
Alabama	Cahaba River pump station, Birmingham		12		
	Mobile River at Government St., Mobile		65		
	Tennessee River at Whitesburg		90		
Arizona	Mineral Creek near Big Dome, 4.8 km south of Ray		130		
Arkansas	Arkansas River near Lock and Dam 3, below Pine Bluff	90			
	Arkansas River at Lock and Dam 6, below Little Rock		12		
	Arkansas River at Lock and Dam 13, below Fort Smith	140			
	Hot Springs Reservoir on Bull Bayou		20		
	Hurricane Creek near Sheridan		15		
	Lake Winona on Alum Fork, Saline River		18		
	North Sylamore Creek near Fifty Six	60			
	Ouachita River near Malvern, below Hot Springs		18		
	White River near Goshen, below Fayetteville	80			
California	Merced River at Happy Isle Bridge near Yosemite				6.0
Connecticut	Naugatuck River at Beacon Falls		22		
Idaho	South Fort Coeur d'Alene River at Smelterville		21		6.8
Illinois	Mississippi River at Chester		12		
	Mississippi River at East St. Louis		16		
	Ohio River at Lock and Dam 53 near Grand Chain		15		
Kentucky	Ohio River at Louisville	70	20		
Louisiana	Calcasieu River near Sulphur		30		
	Cross Lake at Shreveport		16		
	Red River at Moncla		14		
Maine	St. Croix River at Baring		22	890	
Mississippi	Escatawpa River at Moss Point		55		
Missouri	Mississippi River at Cape Girardeau		20		
	Mississippi River at Cottonwood Pt.		15		
	Missouri River at St. Joseph		14		
	Missouri River at Lexington		25		
Nebraska	Big Blue River at Barneston		40		
North Carolina	Cape Fear River at Lock 1 near Kelly	60			
	Catawba River near Charlotte	60			
North Dakota	Red River of the North, below Fargo		26		
Oklahoma	Carney River near Ochelata		12		
	Kiamichi River near Big Cedar			84	

(continued)

TABLE 7.13 (continued)

State	Location	Metal concentration (ppb)			
		As	Cd	Pb	Hg
Pennsylvania	Lackawanna River at Old Forge		32		
	Spring Brook Reservoir near Wilkes-Barre		27		
	Tulpehocken Creek at Blue Marsh damsite near Reading	60			
	West Branch Susquehanna River at Renovo			55	
	Wolf Creek Reservoir near Pottsville		11		
South Carolina	Sugar Creek near Ft. Mill	1100			
South Dakota	James River below Huron		14		
	James River near Mitchell		12		
Tennessee	Watauga River near Johnson City	60			

[a] Many of the locations at which the limit was exceeded are not points where water is withdrawn for public supplies.

Source: Adapted from Anonymous, 1971, Table 2, p. 175. Reprinted by permission of the publisher.

85.1% to 95.8% of the cadmium content of the creek water is in soluble form. Coarse particles (>0.15 μm) accounted for 3.5% to 8.9% of the cadmium, and colloidal particles between 0.01 and 0.15 μm accounted for 0.5% to 6.0%. The coarse and colloid particles contained 13 to 24 ppm and 69 to 161 ppm solid cadmium respectively (Perhac, 1972). Approximately 99% of the cadmium transported by the Walker Branch Stream in Oak Ridge, Tennessee, is in soluble form (Table 7.14) (Andren, Lindberg, and Bate, 1975). Tributary

TABLE 7.14. CADMIUM CYCLING BUDGET IN WALKER BRANCH WATERSHED, OAK RIDGE, TENNESSEE, 1974

Month	Rainfall (cm)	Cadmium concentration (ppb)	Wet input (g/ha)	Estimated dry input[a] (g/ha)	Stream output (g/ha)		Watershed retention (%)
					Dissolved	Suspended	
January	24.47	9.6	23.7	<1.11	1.21		95
February	13.47	9.6	12.9	<0.61	0.64	0.004	95
March	19.74	4.3	8.4	<0.40	0.69		92
April	9.01	3.6	3.3	<0.16	0.53		85
May	17.66	4.5	7.8	<0.37	0.27	0.001	97
June	5.10	12.4	6.3	<0.30	0.14		98
July	2.74	6.3	4.5				

[a] Dry deposition input was estimated based on the July aerosol data that the relative contribution of dry deposition to total atmospheric deposition is constant over the six-month period.

Source: Adapted from Andren, Lindberg, and Bate, 1975, Tables 4, 5, 15, and 16, pp. 14, 15, 32, 33.

streams of Lake Cayuga, New York, in rural areas contained between <0.05 and 0.13 ppb cadmium in particulate form and from 0.07 to 1.10 ppb cadmium in soluble form. Regions of the streams in urban areas contained from 0.09 to 0.44 ppb in particulate form and from 0.07 to 0.29 ppb in soluble form (Kubota, Mills, and Oglesby, 1974). The streams contained more cadmium in April and July than in the other spring and summer months; in April the major portion of cadmium was present in particulate matter, whereas in July soluble cadmium made up the greater fraction (Kubota et al., 1974).

Studies of the cadmium concentration in marine environments have usually been carried out in the vicinity of land masses containing suspected pollution sources. Table 7.10 illustrates the variation in concentrations observed in seawater (Fleischer et al., 1974). Recent studies in the tropical northeast Atlantic Ocean between the Canary Islands and the Cape Verde Islands showed cadmium concentrations ranging from 0.07 to 0.71 ppb (mean of 0.11 ppb), with little change in concentration over depths down to 500 m (Riley and Taylor, 1972).

Concentrations in estuaries and bays can be expected to show considerable variation. Surface waters of Monterey Bay, California, contained 0.24, 0.46, and 0.29 ppb cadmium in March, April, and June; the adjacent open water concentration was 0.05 ppb (Knauer and Martin, 1973). The high value for surface waters of the Corpus Christi Bay estuary during September was 78 ppb near the harbor entrance in contrast to 3 ppb in the bay region. For January, the concentrations only varied from 2 to 10 ppb (Holmes, Slade, and McLerran, 1974).

7.3.5 Distribution in Sediments

Examination of the metal content of sediments can be used to estimate levels of the metal in a large regional drainage area (soil and mineral analyses). In particular, such analyses may be used to assess sources of specific metal pollution in the area (Lisk, 1971). A direct relationship need not exist between the sediment concentration of cadmium and the concentration in water at any given instant. In the Qishon-Gadura river system in Israel, the cadmium concentration in sediments varied from 0 to 123 ppm (with the higher values occurring in the industrial regions of the valley), although the concentration of cadmium in the water was below the detection limit (<0.01 ppm) at all times. It is suggested that the high pH of these waterways (pH 9 to 11 in the contaminated areas) serves to precipitate the cadmium compounds and that any water pollution controls which would lower pH values might lead to increasing solubilization of the cadmium compounds with their subsequent release into Haifa Bay (Kronfeld and Navrot, 1974).

Analyses of sediment cores from lakes and rivers can provide estimates of mineral composition in precultural times and provide information on rates of deposition of heavy metals. Cadmium concentration declined somewhat with depth in the sediments of three of six Wisconsin lakes; the surface concentrations of cadmium were essentially the same (2.1 to 4.6 ppm). For example, in Lake Mendota the 0- to 5-cm sample contained 4.4 ppm cadmium, while below 50 cm, 2.7 ppm cadmium was found (Iskandar and Keeney, 1974). The content of cadmium was below the limit of detection (1 ppm) in analyses of cores from the Great March of Lewes, Delaware (Strom and Biggs, 1972).

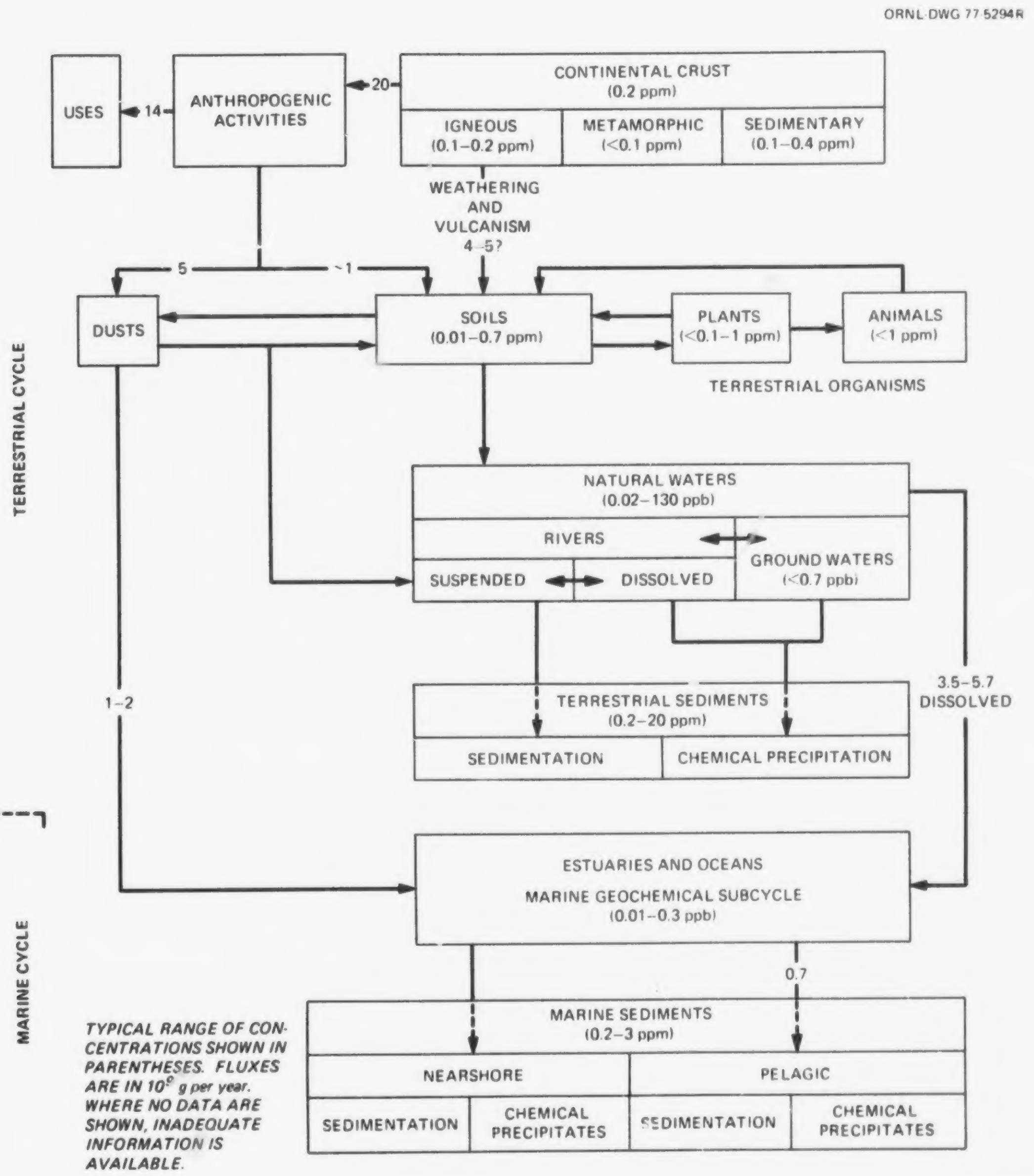
As would be expected, analyses of sediments in the vicinity of pollution sources indicate that considerable quantities of cadmium are deposited. Foundry Cove, New York, which received effluent wastes containing cadmium, nickel, and zinc from a battery manufacturer, contained 39,000 ppm cadmium at the pollution outfall and 1930 ppm at 90 m below the outfall. These findings represent extreme values. Cadmium concentration at the outfall increased with particle size of the sediment, but only 1.3% of the cadmium was isotopically exchangeable; at the 90-m site approximately 74% was exchangeable. These data suggest that cadmium sediments at the outfall may be present in biota or in such insoluble forms as $CdCO_3$; at the 90-m site the cadmium was adsorbed to either clay or organic materials (Bondietti et al., 1974). Studies in the Gulf of Finland gave values for sediments ranging from 0.17 to 1.9 ppm cadmium (dry weight); when expressed per unit non-ash dry weight, unpolluted areas gave values between 4 and 10 ppm, whereas polluted areas had between 70 and 130 ppm (Jaakkola, Takahashi, and Miettinen, 1973). Cadmium levels in San Francisco Bay were 1.22 ± 0.99 ppm for 1- to 7-cm cores and 0.93 ± 0.75 ppm for core depths of 7 to 10 cm (dry weight basis) (Moyer and Budinger, 1974). These authors estimated the "global mean value" for sediments to be about 0.5 ppm cadmium. Santa Barbara Basin cores contained cadmium in concentrations of about 1 to 2 ppm for all depths down to 20 cm (Bruland et al., 1974).

7.4 ENVIRONMENTAL FATE

Distribution and levels of cadmium in air, water, and land were discussed in Section 7.3. A variety of chemical, biological, and physical processes transport cadmium among environmental media with the residence time in a particular medium depending on the extent of these processes. In addition, the biosphere can be considered a medium which can accumulate and concentrate cadmium. Figure 7.6 illustrates the interrelationships among these media and the various processes involved. Such a diagram does not stress sources of cadmium in the environment.

7.4.1 Mobility and Persistence in Air

Little information was found on the residence time of cadmium in air. The observation that concentrations of cadmium in plants and in soils decrease with distance from the point source of pollution suggests that removal does occur either by direct fallout of the dust (or of the CdO) or through removal by precipitation. Distance of travel and persistence in air depend on wind direction and velocity, mixing depth, and stability of the atmosphere. Buchauer (as cited in Friberg et al., 1974) found increased cadmium levels in soil at distances up to 20 km from a zinc smelter. A "dispersion factor" which was determined appears to explain the seasonal distribution of total suspended particles and trace metals in the air over New York City. The February to April cadmium concentration in Manhattan is about 2 to 6 ng/m³, whereas the summer concentration is about 10 to 14 ng/m³; these figures correlate with a lower dispersion factor, the major component of which appears to be the decrease in atmospheric mixing in the summer (Kleinman, Kneip, and Eisenbud, 1974).


ORNL-DWG 77-5294R
USES
14
ANTHROPOGENIC ACTIVITIES
20
CONTINENTAL CRUST (0.2 ppm)
IGNEOUS (0.1–0.2 ppm)
METAMORPHIC (<0.1 ppm)
SEDIMENTARY (0.1–0.4 ppm)
WEATHERING AND VULCANISM 4–5?
5
~1
DUSTS
SOILS (0.01–0.7 ppm)
PLANTS (<0.1–1 ppm)
ANIMALS (<1 ppm)
TERRESTRIAL ORGANISMS
TERRESTRIAL CYCLE
NATURAL WATERS (0.02–130 ppb)
RIVERS
SUSPENDED
DISSOLVED
GROUND WATERS (<0.7 ppb)
1–2
3.5–5.7 DISSOLVED
TERRESTRIAL SEDIMENTS (0.2–20 ppm)
SEDIMENTATION
CHEMICAL PRECIPITATION
ESTUARIES AND OCEANS
MARINE GEOCHEMICAL SUBCYCLE (0.01–0.3 ppb)
0.7
MARINE CYCLE
MARINE SEDIMENTS (0.2–3 ppm)
NEARSHORE
PELAGIC
SEDIMENTATION
CHEMICAL PRECIPITATES
SEDIMENTATION
CHEMICAL PRECIPITATES

TYPICAL RANGE OF CONCENTRATIONS SHOWN IN PARENTHESES. FLUXES ARE IN 10^9 g per year. WHERE NO DATA ARE SHOWN, INADEQUATE INFORMATION IS AVAILABLE.

Figure 7.6. Surface geochemical cycle of cadmium. Source: Adapted from Eaton, 1974, Figure 1, p. 63. Reprinted by permission of the publisher.

Cadmium-109 has been used as a high-altitude tracer. During the period from 1964 to 1968, amounts in precipitation peaked in spring and in late summer to early fall (Ezemenari and Prescott, 1970). Although this pattern indicates that rainfall removes cadmium from air, few quantitative estimates of fallout from low-altitude contamination were found. In the Walker Branch watershed area, the cadmium budget was determined for the first six months of 1974 (Table 7.14). The wet fall input was considerably greater than the dry fall input, and for the six-month period the watershed retained about 95% of atmospheric input (Andren et al., 1974; Andren, Lindberg, and Bate, 1975).

7.4.2 Mobility and Persistence in Soil

The cadmium content of soils varies considerably, depending on soil type as well as the nature of pollution emanating from nearby sources. For most soils not associated with landfill uses, cadmium removal from air by dust fall or precipitation and cadmium added by fertilizer application (Sections 4 and 7.3.3) or sewage sludge (Section 7.5) are the major sources of soil cadmium. Although few direct data were found, water from rivers or lakes could add cadmium to soils on flood plains or in areas of extensive irrigation. In Japan, paddy soil analyses and distribution studies suggested that cadmium was transported in the form of suspended particles in irrigation water obtained from a stream passing through a mining area (Yamagata and Shiegematsu, 1970).

The mobility and persistence of cadmium in soils depend on chemical form and the physical and chemical characteristics of the soil. Cadmium is considered a slowly mobile element because it readily forms insoluble compounds under oxidizing conditions. In addition, the form deposited from the air would probably be either CdO or cadmium in larger particulate form. The observation that the cadmium concentration is highest in the surface layer of soil (Section 7.3.3) supports the contention that cadmium is relatively immobile.

An important factor determining the form of cadmium in soils is pH. At low pH values, cadmium compounds are more soluble and, thus, cadmium is available for plant uptake. At higher values ($pH > 8$), cadmium in solution can undergo hydrolysis to form $CdOH^+$ (Hahne and Kroontje, 1973); however, if any carbonate is available, the insoluble $CdCO_3$ will precipitate. Similarly, CdO and CdS are relatively insoluble compounds that decrease the availability of cadmium in soil. Thus, liming of the soil results in the formation of insoluble heavy-metal compounds (Section 2.3.2.1). However, under favorable soil conditions inorganic sulfur compounds can be oxidized by microorganisms (Alexander, 1961). Microbiological oxidation is far more rapid than chemical changes at near-optimum moisture and temperature conditions. Thus, sulfides in soil can be made available for plant uptake when attacked by microorganisms (Fassett, 1972). No specific data could be found for cadmium release by sulfide oxidation.

Another mechanism for cadmium retention in soil is through ion exchange reactions with clay and organic matter. Lagerwerff and Brower (1972) studied exchange adsorption characteristics of different clays and reported that "the exchange of Cd^{2+} in Al^{3+}- and Cd^{2+}-treated soils was normal in that the adsorption was greater in the presence of Ca^{2+} than of Al^{3+}, and decreased with increasing concentrations of $AlCl_3$ and $CaCl_2$." Bondietti et al. (1974) reported that cadmium adsorption by clays and sesquioxides (iron and aluminum oxides, common constituents of soils) increases with pH. Humic acids of the soil were also reported to adsorb or complex cadmium.

Clay and organic fractions thus hold cadmium in an exchangeable state which ultimately may be removed by plants. Wentink and Etzel (1972) referred to Grim's list of five factors affecting replaceability of cations: (1) cation concentration, (2) population of exchange positions, (3) nature of accompanying anion, (4) nature of cation, and (5) nature of clay material.

The observation that the level of heavy metals in soil decreases in open habitats after closure of the polluting smelter shows that various processes act to dissipate contaminants. The time for 50% depletion of cadmium was 1.98 and 2.38 years for two bare-surface sites and 11 years for a grass-covered area; vegetation growth in previously bare areas is expected to increase the time required to reach background levels (Roberts and Goodman, 1974). Processes causing this depletion were not discussed, but combinations of wind, erosion, runoff, and leaching through soil are possibilities.

Cadmium leached from soils by water runoff may be carried to regional groundwater and eventually to rivers. The seepage of cadmium-containing plating waste from disposal basins to groundwater has been documented in Long Island, New York. Perlmutter and Lieber (1970) reviewed the movement and spread of plating wastes derived from chemical solutions used in anodizing and other metal-plating processes. Intermittently since 1941, plating wastes containing cadmium, hexavalent chromium, and other heavy metals have seeped down from disposal basins through the zone of aeration in the soil and into the zone of saturation in the upper glacial aquifer directly beneath the basins. The seepage formed a plume of contaminated water about 13,115 m (4300 ft) long and as much as 3050 m (1000 ft) wide and 213.5 m (70 ft) thick. The plume extended down gradient to the headwaters of Massapequa Creek, which formed a natural drain for part of the contaminated water, while the remainder moved slowly down gradient as underflow beneath and parallel to the stream channel. After reviewing the significant geochemical features of the plume from 1949 to 1962, Perlmutter and Lieber (1970) concluded that variations in the quality of the water in the plume down gradient from the disposal basins may be partly attributable to differences in the age and history of the contaminated water in different parts of the plume. Cadmium concentrations were found to vary in different areas of the plume; the authors suggested that variations in concentrations were probably due to changes in the chemical character of treated effluent and also to the influence of hydrological and geochemical factors. Concentrations of cadmium were generally less than 1 ppm at most test wells. However, in 1964, samples from one test site showed 10 ppm cadmium. This concentration was determined in a zone near the center of the principal path of flow from the disposal basins. Cadmium lixiviation from buried mine tailings was demonstrated in the Canyon Creek Basin near Wallace, Idaho (Mink, Williams, and Wallace, 1972).

7.4.3 Mobility and Persistence in Water and Sediments

Cadmium in the dissolved state in water can be expected to undergo reactions which depend on other constituents of the water. Solubilities of cadmium compounds are known and the effects of pH and bicarbonate concentration have been reported (Hem, 1972) (Section 2.3.2.1). Gardiner (1974) stated that concentrations of soluble cadmium in fresh water are lower than the maximum predicted from the solubility data for cadmium carbonate, the least soluble cadmium salt. Clearly, adsorption to solids is implicated in removal of cadmium from waters. From the experimental data, Gardiner (1974) concluded that adsorption onto mud is rapid and can give concentration factors of 5000 to 50,000, depending upon type of solid, pH, and water hardness. Humic material in the water seems to be the major component responsible for this adsorption. Martin (1970) suggested that abundant molted copepod exoskeletons (insoluble chitin) can adsorb considerable amounts of heavy metals from natural waters. Judging from elemental analysis of stream water, rain, and soils, the source of cadmium dissolved in the Walker Branch Stream, Oak Ridge, Tennessee (Table 7.14) appears to be precipitation (Andren, Lindberg, and Bate, 1975).

The fate of any cadmium precipitate or adsorbed cadmium in water depends on the ability of the particles to settle. In Corpus Christi Bay, Texas, the water stagnation in summer — along with the inflow of cadmium pollution — increases the cadmium concentration until CdS is precipitated in the anaerobic depths of the harbor. With cold air fronts, water mixing occurs and circulation increases between the bay and harbor; this circulation oxygenates the water, which results in desorption from precipitates with transportation of the metals into the bay (Holmes, Slade, and McLerran, 1974). De Groot and Allersma (1973) found that metals remain fixed to the suspended matter in river water as long as the water does not mix with the sea. Mobilization can occur through decomposition of organic matter releasing organometallic complexes and yielding less contaminated sediments in the lower regions of deltas.

Data are limited on the fate of cadmium in sediments of bodies of water. Certainly, a considerable amount is eventually carried to the sea with river silt and suspended matter. Deep-sea sediments contain from 40 to 580 ppb cadmium (average 225 ppb) (Aston et al., 1972). Table 7.11 presents data on the amount of cadmium flow occurring in several rivers. Ultimately, most of this cadmium enters the sea; cadmium concentration in the seawater is quite low (about 0.1 ppb) because most cadmium is deposited in deep-sea sediments (Fleischer et al., 1974).

7.5 WASTE MANAGEMENT

Disposal of various domestic wastes is an acute problem due to increasing industrialization and urbanization. This section deals with treatment of certain waste materials that contain cadmium.

Major sources of industrial liquid and solid wastes which contain considerable cadmium are (1) the electroplating industry (liquid slurry and sludge), (2) nickel-cadmium battery manufacturers (sludge), (3) paint manufacturers (sludge), and (4) paint in old containers (residues to municipal dumps) (Ottinger et al., 1973). In addition, cadmium dusts and fumes from extraction, refining, and processing industries constitute a major source of atmospheric cadmium.

Prevention of cadmium emissions to the air is accomplished through the use of electrostatic precipitators, bag filters, and cyclones (Goeller and Flora, 1973). The efficiency for capture of particulate emissions is expected to increase as technology advances in these areas (Adams, 1974; Frey, 1974).

The usual disposal method for concentrated sludges or wastes involves coagulation with lime, sedimentation, and then filtration through sand. These residues are then dried and can be placed in approved chemical landfills (Ottinger et al., 1973). Depending on cadmium form and concentration and on technological developments, wastes could theoretically be used for cadmium recovery, although at present no cadmium is removed for commercial use.

Conventional sewage treatment produces two types of discharge: a treated wastewater effluent and a concentrated sludge. The wastewater is discharged into bodies of water, but sludge disposal usually involves either application to land or incineration (Yost et al., 1973). Application of sewage sludge to cropland and other lands is based on the concepts that (1) microorganisms will degrade the organic constituents, (2) soil particles will retain many of the elements of the sludge, (3) soil will serve as a filtering system to remove solids from the sludge, and (4) nutrients deposited in soil will be available for plant absorption. One of the drawbacks, however, is the heavy-metal concentration in sludge — 600 to 900 ppm dry weight in industrial areas and 10 to 50 ppm in rural communities (Yost et al., 1973). Heavy-metal composition of sludge varies considerably; for instance, samples of sludge from Michigan ranged from 2 to 1100 ppm with a median of 12 ppm and an average of 74 ppm (Page, 1974). The ranges of metal content in digested sewage sludge are presented in Table 7.15; average concentrations of metals in raw sludge are shown in Table 7.16. The concentration of cadmium in several industrial sludges is given in Table 7.17. Because plants absorb heavy metals (Section 4), increased levels in plants are a potential hazard to humans (Stenström and Lönsjö, 1974). It has been shown that when plants are grown on sewage sludge their cadmium content may be increased (Sections 4.2.2.1.1 and 4.2.2.1.2; see also, however, Section 7.3.3). Chaney (1973) pointed out that toxic metals added to soils are not a hazard to the food chain until they have entered an edible part of a plant. However, some direct ingestion of soil containing large amounts of metals may become a hazard to animals grazing on sludge- or effluent-treated sites.

In a recent literature review, Page (1974) cited information which suggests that cadmium applied in sludge is not mobile and does not move significantly beyond the depth of tillage. Practices employing deeper tillage may allow for greater amounts of sludge to be applied per unit area.

TABLE 7.15. RANGE OF METAL CONTENTS IN DIGESTED SEWAGE SLUDGE

Element	Observed range (ppm)
Zinc	500-50,000
Copper	250-17,000
Nickel	25-8,000
Cadmium	5-2,000 0.1-40% of Zn
Boron	15-1,000
Lead	100-10,000
Mercury	<1-10

Source: Adapted from Chaney, 1973, Table 1, p. 130.

TABLE 7.16. AVERAGE CONCENTRATIONS OF METALS IN RAW SLUDGE

Metal	Metal concentration (mg/kg dry wt)				
	Arithmetic mean	Standard deviation	Geometric mean	Standard deviation[a]	Median 50% value
Silver	490	370	355	2.51	<100
Boron	880	410	775	1.67	805
Cadmium	30	15	27	1.53	20
Calcium	13,800	7,830	11,700	1.82	13,900
Chromium	1,370	1,400	940	2.75	750
Cobalt	700	770	410	2.32	240
Copper	860	550	740	1.67	660
Mercury	15	23	8.2	2.54	5.5
Manganese	1,310	2,860	460	3.32	200
Nickel	580	540	420	2.12	335
Lead	1,380	775	1,150	1.95	1,150
Strontium	190	75	175	1.45	<100
Zinc	1,960	1,000	1,740	1.66	1,880

[a] Standard deviation of the geometric mean is a ratio and has no units.

Source: Adapted from Salotto, Grossman, and Ferrell, 1974, Table 5, p. 52.

TABLE 7.17. CONCENTRATION OF CADMIUM IN SEVERAL INDUSTRIAL SLUDGES

Industry and/or type of sludge	Cadmium concentration (ppm)
Electroplating, dried sludge	1000-5000
Electronics manufacturing	10-100
Primary zinc smelters and refiners	
Fresh anode sludge	12
Old anode sludge	1400
Secondary lead smelters	
Scrubber sludge	340
Wastewater treatment sludge	10
Secondary copper smelter, water treatment sludge	10
Primary lead smelter and refiners, sinter scrubber sludge	900
Primary copper plants, sludge from lagoon receiving acid plant blowdown	180

Source: Compiled from National Commission on Water Quality, 1976, and Calspan Corporation, 1975.

Application of sludge containing low concentrations of cadmium can be expected to enrich the upper regions of the soil over a relatively short period.

Cadmium removal from water effluents can be considered from the viewpoint of industrial waters and municipal wastewaters. Treatment for cadmium removal from various industrial wastewaters usually involves either chemical precipitation at high pH with lime or use of ion exchange procedures. Although chemical precipitation is the more common procedure, the use of ion exchange may become feasible when the product recovery value of cadmium increases (Watson, 1973). Aqueous wastes from many electroplating plants are cyanide solutions $[Cd(CN)_4{}^{2-}]$ for which improved methods of removal are needed. Use of quaternary amines followed by organic solvent extraction is one possible removal system (Moore, Groenier, and Bayless, 1974).

Treated sewage water effluents are discharged into natural waters or are filtered through soils. Lehman and Wilson (1971) reported that concentrations of cadmium and several other heavy metals were "effectively reduced during percolation through about 8 feet of calcareous soil material contained in lysimeters." Cadmium was deposited near the surface of the lysimeter. Field plot studies indicated that the soil can become saturated with several elements after three years of intermittent flooding; however, cadmium was not tested in these experiments.

Urban area studies analyzing patterns of wastewater loading are necessary to identify and estimate the relative importance of each source and to determine trends in the release of materials. Although heavy metals were not individually characterized, studies for the San Francisco Bay region indicated that municipal rather than industrial discharges may be the major source of heavy-metal pollution (Breslaw, 1974). Necessary improvements to the components of the wastewater pollution control system depend on the major polluting source for the locale.

Major inputs of pollutants into municipal discharge have been listed as (1) wastewater treatment effluents, (2) direct domestic and industrial wastewaters, (3) combined sewer overflow, and (4) storm runoff from urban, agricultural, and other rural areas (Warren and Bewtra, 1974). These authors constructed a computer model to study the effects of time-variable pollutant loads on stream quality.

The Hyperion Treatment Plant in Los Angeles discharges primary effluents, secondary effluents, and digested sludge into the Pacific Ocean. Approximately 80% of the cadmium from the primary effluent (28 ppm) was particulate (sizes greater than 0.22 μm), while only about 10% of the secondary effluent (10 ppm) was particulate (Chen et al., 1974). The settling process in secondary treatment produced a 64% decrease in total concentration of cadmium, but the soluble cadmium concentration increased from 2.7 ppb in the primary effluent to 9.0 ppb in the secondary effluent. The distribution of cadmium in particulate material of the primary effluent was 34% in the 2- to 8-μm range, 58% in the 8- to 44-μm range, and 10% in the >44-μm range. For secondary effluents, the corresponding percentages were 32%, 53%, and 15%. The 0.2- to 0.8-μm particles contained a higher fraction of cadmium than the larger particles, perhaps due to the relative surface available for adsorption.

The electroplating industry releases about 30 kg (65 lb) of cadmium daily into the sewage waters of New York; however, analysis of water influents at 12 major water treatment plants showed a content of about 73 kg (160 lb) cadmium per day (from 0.005 to 0.050 ppm). This analysis indicates that other sources of cadmium discharge exist besides the electroplating industry. Residential wastewater contained from 0.001 to 0.007 ppm cadmium; wastewater from other industries varied from 0.006 (fat rendering) to 0.134 ppm (laundry), and surface runoff contained 0.025 ppm cadmium. Thus, electroplaters accounted for 33% of the cadmium received in influents at water plants, runoff accounted for 12%, other industries accounted for 6%, and residential runoff accounted for 49% of total cadmium. Water released into New York Harbor comes from treatment plant effluents, storm runoff from sewer overflow and storm sewers, and untreated wastewaters from areas not served by treatment plants (about 43, 50, and 27 kg/day cadmium respectively). Cadmium content of sludge dumped at the sludge dumping grounds varied from 0.4 to 9.8 ppm. Concentration of cadmium in harbor waters varied between 0.0008 and 0.0055 ppm, compared with open Atlantic Ocean values of 0.0023 ppm. In New York City, then, nonindustrial sources of water pollution are significant and complicate efforts to eliminate all discharges of metal. As a result, release of hazardous metals into societal flow may have to be severely restricted (Klein et al., 1974).

The Environmental Protection Agency has proposed guidelines for utilization and disposal of sludges (*Federal Register*, 1976). The Metropolitan Sanitary District of Greater Chicago (Lue-Hing et al., 1977) succeeded by ordinance in reducing the cadmium content of sludge at its Calumet treatment plant by 72% over a period of five years. Clearly, it may become necessary generally to restrict release of hazardous trace metals into societal flow (Klein et al., 1974).

Another method used to treat sewage secondary effluent is the use of stabilization ponds. Sediments from these ponds and the aquatic vegetation in them represent potential sinks for accumulation of contaminating elements. In a Michigan study, concentrations of chromium, copper, iron, manganese, and zinc were reduced in these ponds; however, cadmium, cobalt, and nickel were not affected (Bulthuis, Craig, and McNabb, 1973).

SECTION 7

REFERENCES

1. Abdullah, M. I., L. G. Royle, and A. W. Morris. 1972. Heavy Metal Concentration in Coastal Waters. Nature (Great Britain) 235:158-160.

2. Adams, R. L. 1974. Designing a Filter System to Meet Specified Efficiency and Emission Levels. J. Air Pollut. Control Assoc. 24(12):1168-1170.

3. Alexander, M. 1961. Introduction to Soil Microbiology. John Wiley and Sons, Inc., New York. 472 pp.

4. Andren, A. W., B. G. Blaylock, E. A. Bondietti, C. F. Francis, S. G. Hildebrand, J. W. Huckabee, D. R. Jackson, S. E. Lindberg, F. H. Sweeton, R. I. Van Hook, and A. P. Watson. 1974. Ecological Research. In: Ecology and Analysis of Trace Contaminants. ORNL/NSF/EATC-11, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 61-79.

5. Andren, A. W., S. E. Lindberg, and L. C. Bate. 1975. Atmospheric Input and Geochemical Cycling of Selected Trace Elements in Walker Branch Watershed. ORNL/NSF/EATC-13, Oak Ridge National Laboratory, Oak Ridge, Tenn. 68 pp.

6. Anonymous. 1971. USGS Completes Nationwide Reconnaissance of Metals in Streams. Water & Sewage Works 118(6):174-175.

7. Aston, S. R., R. Chester, A. Griffiths, and J. P. Riley. 1972. Distribution of Cadmium in North Atlantic Deep-Sea Sediments. Nature (Great Britain) 239:393.

8. Baes, C. F., Jr. 1973. The Properties of Cadmium. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 29-60.

9. Bondietti, E. A., R. M. Perhac, F. H. Sweeton, and T. Tamura. 1974. Toxic Metals in Sediments. In: Ecology and Analysis of Trace Contaminants. ORNL/NSF/EATC-6, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 176-185.

10. Breslaw, J. A. 1974. Sources and Trends in Waste Water Loadings to the San Francisco Bay Region. Water Resour. Res. 10(6):1085-1089.

11. Bruland, K. W., K. Bertine, M. Koide, and E. D. Goldberg. 1974. History of Metal Pollution in Southern California Coastal Zone. Environ. Sci. Technol. 8(3):425-432.

12. Bulthuis, D. A., J. R. Craig, C. D. McNabb. 1974. Metal Dynamics in Municipal Stabilization Ponds. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 7:127-135.

13. Butterworth, J., P. Lester, and G. Nickless. 1972. Distribution of Heavy Metals in the Severn Estuary. Mar. Pollut. Bull. (Great Britain) 3(5):72-74.

14. Calspan Corporation. 1977. Assessment of Industrial Hazardous Waste Practices in the Metal Smelting and Refining Industry, Vol. II. U.S. Environmental Protection Agency, Washington, D.C. 309 pp.

15. Chaney, R. L. 1973. Crop and Food Chain Effects of Toxic Elements in Sludges and Effluents. In: Proceedings of the Joint Conference on Recycling Municipal Sludges and Effluents on Land. U.S. Environmental Protection Agency, U.S. Department of Agriculture, and National Association of State Universities and Land-Grant Colleges. pp. 129-141.

16. Chen, K. Y., C. S. Young, T. K. Jan, and N. Rohatgi. 1974. Trace Metals in Wastewater Effluents. J. Water Pollut. Control Fed. 46(12):2663-2675.

17. Creason, J. P., O. McNulty, L. T. Heiderscheit, D. H. Swanson, and R. W. Buechley. 1972. Roadside Gradients in Atmospheric Concentrations of Cadmium, Lead, and Zinc. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 5:129-142.

18. de Groot, A. J., and E. Allersma. 1973. Field Observations on the Transport of Heavy Metals in Sediments (presented at Conference on Heavy Metals in the Aquatic Environment, Nashville, Tenn., December 4-7, 1973). 14 pp.

19. De Koning, H. W. 1974. Lead and Cadmium Contamination in the Area Immediately Surrounding a Lead Smelter. Water Air Soil Pollut. (Netherlands) 3:63-70.

20. Doolan, K. J., and L. E. Smythe. 1973. Cadmium Content of Some New South Wales Waters. Search (Australia) 4(5):162-163.

21. Durum, W. H., and J. D. Hem. 1972. An Overview of Trace Element Distribution Patterns in Water. Ann. N.Y. Acad. Sci. 199:26-36.

22. Eaton, A. D. 1974. Marine Geochemistry of Cadmium. Ph.D. Dissertation. Harvard University, Cambridge, Mass. 231 pp.

23. Ezemenari, F.R.C., and J. R. Prescott. 1970. Autumnal Peak in ^{109}Cd Fallout in Precipitation at Calgary, Canada. J. Geophys. Res. 75(27):5271-5275.

24. Fassett, D. W. 1972. Cadmium. In: Metallic Contaminants and Human Health, D.H.K. Lee, ed. Academic Press, New York. pp. 97-124.

25. Federal Register. 1976. 41:22532-22536.

26. Fleischer, M., A. F. Sarofim, D. W. Fassett, P. Hammond, H. T. Shacklette, I.C.T. Nisbet, and S. Epstein. 1974. Environmental Impact of Cadmium: A Review by the Panel on Hazardous Trace Substances. Environ. Health Perspect. 7:253-323.

27. Frey, R. E. 1974. Types of Fabric Filter Installations. J. Air Pollut. Control Assoc. 24(12):1148-1150.

28. Friberg, L., M. Piscator, G. F. Nordberg, and T. Kjellström. 1974. Cadmium in the Environment, 2nd ed. CRC Press, Cleveland, Ohio. 248 pp.

29. Gardiner, J. 1974. The Chemistry of Cadmium in Natural Water: II. The Adsorption of Cadmium on River Muds and Naturally Occurring Solids. Water Res. (Great Britain) 8:157-164.

30. Goeller, H. E., and H. B. Flora II. 1973. Abatement Problems and Practices for Zinc and Cadmium. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 135-202.

31. Goeller, H. E., E. C. Hise, and H. B. Flora II. 1973. Societal Flow of Zinc and Cadmium. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 61-134.

32. Goldberg, A. J. 1973. A Survey of Emissions and Controls for "Hazardous" and Other Pollutants. EPA/R4-73/021, U.S. Environmental Protection Agency, Washington, D.C. 165 pp.

33. Gordon, G. E., W. H. Zoller, and E. S. Gladney. 1974. Abnormally Enriched Trace Elements in the Atmosphere. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 7:167-174.

34. Hahne, H.C.H., and W. Kroontje. 1973. Significance of pH and Chloride Concentration on Behavior of Heavy Metal Pollutants: Mercury(II), Cadmium(II), Zinc(II), and Lead(II). J. Environ. Qual. 2(4):444-450.

35. Harrison, P. R., and J. W. Winchester. 1971. Area-wide Distribution of Lead, Copper, and Cadmium in Air Particulates from Chicago and Northwest Indiana. Atmos. Environ. (Great Britain) 5:863-880.

36. Heindl, R. A. 1970. Cadmium. In: Mineral Facts and Problems. U.S. Department of the Interior, Washington, D.C. pp. 515-526.

37. Hem, J. D. 1972. Chemistry and Occurrence of Cadmium and Zinc in Surface Water and Groundwater. Water Resour. Res. 8(3):661-679.

38. Holmes, C. W., E. A. Slade, and C. J. McLerran. 1974. Migration and Redistribution of Zinc and Cadmium in Marine Estuarine System. Environ. Sci. Technol. 8(3):255-259.

39. Hunt, W. F., Jr., C. Pinkerton, O. McNulty, and J. Creason. 1971. A Study in Trace Element Pollution of Air in 77 Midwestern Cities. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 4:56-68.

40. Iskandar, I. K., and D. R. Keeney. 1974. Concentration of Heavy Metals in Sediment Cores from Selected Wisconsin Lakes. Environ. Sci. Technol. 8(2):165-170.

41. Jaakkola, T., H. Takahashi, and J. K. Miettinen. 1973. Cadmium Content in Sea Water, Bottom Sediment, Fish, Lichen, and Elk in Finland. Environ. Qual. Saf. 2:230-237.

42. John, M. K., H. H. Chuah, and C. J. VanLaerhoven. 1972. Cadmium Contamination of Soil and Its Uptake by Oats. Environ. Sci. Technol. 6(6):555-557.

43. Klein, D. H. 1972. Mercury and Other Metals in Urban Soils. Environ. Sci. Technol. 6(6):560-562.

44. Klein, L. A., M. Lang, N. Nash, and S. K. Kirschner. 1974. Sources of Metals in New York City Wastewater. J. Water Pollut. Control Fed. 46(12):2653-2662.

45. Kleinman, M. T., T. J. Kneip, and M. Eisenbud. 1974. Meteorological Influences on Airborne Trace Metals and Suspended Particulates. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 7:161-166.

46. Knauer, G. A., and J. H. Martin. 1973. Seasonal Variations of Cadmium, Copper, Manganese, Lead, and Zinc in Water and Phytoplankton in Monterey Bay, California. Limnol. Oceanogr. 18(4):597-604.

47. Kneip, T. J., M. Eisenbud, C. D. Strehlow, and P. C. Freudenthal. 1970. Airborne Particulates in New York City. J. Air Pollut. Control Assoc. 20(3):144-149.

48. Kobayashi, J. 1972. Air and Water Pollution by Cadmium, Lead and Zinc Attributed to the Largest Zinc Refinery in Japan. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 5:117-128.

49. Kronfeld, J., and J. Navrot. 1974. Transition Metal Contamination in the Qishon River System, Israel. Environ. Pollut. (Great Britain) 6(4):281-288.

50. Kubota, J., E. L. Mills, and R. T. Oglesby. 1974. Lead, Cd, Zn, Cu, and Co in Streams and Lake Waters of Cayuga Lake Basin, New York. Environ. Sci. Technol. 8(3):243-248.

51. Lagerwerff, J. V., and D. L. Brower. 1972. Exchange Adsorption of Trace Quantities of Cadmium in Soils Treated with Chlorides of Aluminum, Calcium, and Sodium. Soil Sci. Soc. Am. Proc. 36:734-737.

52. Lagerwerff, J. V., D. L. Brower, and G. T. Biersdorf. 1973. Accumulation of Cadmium, Copper, Lead and Zinc in Soil and Vegetation in the Proximity of a Smelter. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 6:71-78.

53. Lagerwerff, J. V., and A. W. Specht. 1970. Contamination of Roadside Soil and Vegetation with Cadmium, Nickel, Lead, and Zinc. Environ. Sci. Technol. 4(7):583-586.

54. Lee, R. E., Jr., R. K. Patterson, and J. Wagman. 1968. Particle-Size Distribution of Metal Components in Urban Air. Environ. Sci. Technol. 2(4):288-290.

55. Lehman, G. S., and L. G. Wilson. 1971. Trace Element Removal from Sewage Effluent by Soil Filtration. Water Resour. Res. 7(1):90-99.

56. Lisk, D. J. 1971. Ecological Aspects of Metals. N.Y. State J. Med. 71(21):2541-2555.

57. Lue-Hing, C., T. D. Hinesly, J. R. Peterson, and D. R. Zenz. 1977. Heavy Metal Uptake and Control Strategies Associated with Sewage Sludge Fertilized Crops. Report No. 77-4, Department of Research and Development, Metropolitan Sanitary District of Greater Chicago.

58. Marowsky, G., and K. H. Wedepohl. 1971. General Trends in the Behavior of Cadmium, Mercury, Thallium and Bismuth in Some Major Rock Forming Processes. Geochim. Cosmochim. Acta 35:1255-1267.

59. Martin, J. H. 1970. The Possible Transport of Trace Metals via Moulted Copepod Exoskeletons. Limnol. Oceanogr. 15(5):756-761.

60. McCabe, L. J., J. M. Symons, R. D. Lee, and G. G. Robeck. 1970. Survey of Community Water Supply Systems. J. Am. Water Works Assoc. 62:670-687.

61. Mink, L. L., R. E. Williams, and A. T. Wallace. 1972. Effect of Early Day Mining Operations on Present Day Water Quality. Ground Water 10(1):17-26.

62. Moore, F. L., W. S. Groenier, and W. E. Bayless. 1974. Recovery of Toxic Metals from Industrial Effluent Solutions by Solvent Extraction. In: Ecology and Analysis of Trace Contaminants. ORNL/NSF/EATC-11, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 333-340.

63. Moyer, B. R., and T. F. Budinger. 1974. Cadmium Levels in the Shoreline Sediments of San Francisco Bay. Donner Laboratory and Lawrence Berkeley Laboratory, Berkeley, Calif. 37 pp.

64. Natusch, D.F.S., J. R. Wallace, and C. A. Evans, Jr. 1974. Toxic Trace Elements: Preferential Concentration in Respirable Particles. Science 183:202-204.

65. National Commission on Water Quality. 1976. Staff Report to the National Commission on Water Quality. Washington, D.C.

66. Ottinger, R. S., J. L. Blumenthal, D. F. Dal Porto, G. I. Gruben, M. J. Santy, and C. C. Shih. 1973. Recommended Methods of Reduction, Neutralization, Recovery, or Disposal of Hazardous Waste. EPA-670/2-73-053-a, U.S. Environmental Protection Agency, Cincinnati, Ohio. pp. 78-79.

67. Page, A. L. 1974. Fate and Effects of Trace Elements in Sewage Sludge When Applied to Agricultural Lands — A Literature Review Study. EPA-670/2-74-005, U.S. Environmental Protection Agency, Cincinnati, Ohio. 96 pp.

68. Pasternak, K. 1973. The Spreading of Heavy Metals in Flowing Waters in the Region of Occurrence of Natural Deposits and of the Zinc and Lead Industry. Acta Hydrobiol. (Poland) 15(2):145-166.

69. Perhac, R. M. 1972. Distribution of Cd, Co, Cu, Mn, Ni, Pb and Zn in Dissolved and Particulate Solids from Two Streams in Tennessee. J. Hydrol. (Netherlands) 15:177-186.

70. Perlmutter, N. M., and M. Lieber. 1970. Dispersal of Plating Wastes and Sewage Contaminants in Ground Water and Surface Water, South Farmingdale-Massapequa Area, Nassau County, New York. Geological Survey Water-Supply Paper 1879-G, U.S. Department of the Interior, Washington, D.C. 67 pp.

71. Proctor, P. D., G. Kisvarsanyi, E. Garrison, and A. Williams. 1974. Heavy Metal Content of Surface and Ground Waters of the Springfield-Joplin Areas, Missouri. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 7:63-69.

72. Ratsch, H. C. 1974. Heavy-Metal Accumulation in Soil and Vegetation from Smelter Emissions. EPA-660/3-74-012, U.S. Environmental Protection Agency, Corvallis, Ore. 23 pp.

73. Riley, J. P., and D. Taylor. 1972. The Concentrations of Cadmium, Copper, Iron, Manganese, Molybdenum, Nickel, Vanadium, and Zinc in Part of the Tropical North-east Atlantic Ocean. Deep-Sea Res. (Great Britain) 19:307-317.

74. Roberts, T. M., and G. T. Goodman. 1974. The Persistence of Heavy Metals in Soils and Natural Vegetation following Closure of a Smelter. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 7:117-125.

75. Salotto, B. V., E. Grossman, and J. B. Farrell. 1974. Elemental Analysis of Wastewater Sludges from 33 Wastewater Treatment Plants in the United States. In: Pretreatment and Ultimate Disposal of Wastewater Solids. EPA-902/9-74-002, U.S. Environmental Protection Agency, New York. pp. 23-72.

76. Schroeder, H. A., A. P. Nason, I. H. Tipton, and J. J. Balassa. 1967. Essential Trace Elements in Man: Zinc; Relation to Environmental Cadmium. J. Chronic Dis. (Great Britain) 20:179-210.

77. Stenström, T., and H. Lönsjö. 1974. Cadmium Availability to Wheat: A Study with Radioactive Tracers under Field Conditons. Ambio (Norway) 3:87-90.

78. Stenström, T., and M. Vahter. 1974. Cadmium and Lead in Swedish Commercial Fertilizers. Ambio (Norway) 3:91-92.

79. Strom, R. N., and R. B. Biggs. 1972. Trace Metals in Cores from the Great Marsh, Lewes, Delaware. University of Delaware, Newark, Del. 35 pp.

80. Toca, F. M., C. L. Cheever, and C. M. Berry. 1973. Lead and Cadmium Distribution in the Particulate Effluent from a Coal-fired Boiler. Am. Ind. Hyg. Assoc. J. 34(9):396-403.

81. U.S. Environmental Protection Agency. 1973. Air Quality Data for Metals, 1968 and 1969. Research Triangle Park, N.C. pp. 4.9-4.13.

82. Warren, J., and J. K. Bewtra. 1974. A Model to Study the Effects of Time-Variable Pollutant Loads on Stream Quality. Water. Res. (Great Britain) 8:1057-1061.

83. Watson, M. R. 1973. Cadmium Removal from Water. In: Pollution Control in Metal Finishing. Noyes Data Corporation, Park Ridge, N.J. pp. 87-89.

84. Wentink, G. R., and J. E. Etzel. 1972. Removal of Metal Ions by Soil. J. Water Pollut. Control Fed. 44(8):1561-1574.

85. Williams, C. H., and D. J. David. 1973. The Effect of Superphosphate on the Cadmium Content of Soils and Plants. Aust. J. Soil Res. (Australia) 11:43-56.

86. Yamagata, N., and I. Shigematsu. 1970. Cadmium Pollution in Perspective. Koshu Eiseiin Kenkyu Hokoku (Japan) 19:1-27.

87. Yost, K. J., W. Burns, J. E. Christian, F. M. Clikeman, R. B. Jacko, D. R. Masarik, W. W. McFee, A. W. McIntosh, J. E. Newman, R. I. Pietz, and A. M. Zimmer. 1973. The Environmental Flow of Cadmium and Other Trace Metals, Vol. I. Purdue University, West Lafayette, Ind. pp. 27-42.

SECTION 8

ENVIRONMENTAL INTERACTIONS AND THEIR CONSEQUENCES

8.1 SUMMARY

Various processes interact to redistribute cadmium among land, water, and air. Organisms can take up cadmium from these media and, in certain cases, can concentrate it. Because cadmium is a toxic element, it is important to identify foods with a high cadmium content and food chains in which biomagnification of cadmium concentration occurs. A variety of foods have a low, but measurable, cadmium content. In general, the cadmium content of vegetables and grains reflects that in the soil but does not represent the same high magnification found in some neritic organisms (especially oysters). Little comprehensive work has been done on the movement of cadmium in food chains, although concentration and radiocadmium data show that cadmium does move within the various components of an ecosystem.

8.2 ENVIRONMENTAL CYCLING OF CADMIUM

In Section 7 the sources, distribution, and fate of cadmium in air, water, and land were described. In addition to background levels of cadmium from the processes of weathering and extraction from natural deposits, elevated levels result from various mining, refining, and processing steps and from disposal of various municipal wastes. Figure 7.6 illustrates the environmental cycling of cadmium. Fallout of cadmium-containing dust and its removal by rain deposit cadmium on land and in water. Although the residence time in these two media may be longer than in air, the condition is not static. Cadmium can be retained in the soil through adsorption by clay and organic matter. Ion exchange can occur, however, depending on the type and concentration of other ions within any percolating solution. This reaction releases cadmium to the soil solution where it can be leached into groundwater. Similarly, cadmium in soil is available for plant uptake (Section 4.2.1). Wastewater discharges (municipal and industrial), as well as runoff from urban and rural land masses, add cadmium to surface waters. The cadmium in these waters can be precipitated and deposited in sediments. Thus, there is a continuing anthropogenic input of cadmium into the environment with a subsequent complex cycling of the element among environmental media.

8.3 FOOD CHAINS

8.3.1 Cadmium in Foods

The effects on the environment, particularly the biota, from increased levels of cadmium — and ultimately the effects on man — are a major concern. Human exposure to cadmium occurs through food, air, and water. A reasonable estimate of the average total daily cadmium intake by humans is about 50 μg, the major part of which is probably contributed by food (Friberg et al., 1974). Figure 8.1 identifies cadmium concentrations in surface waters,

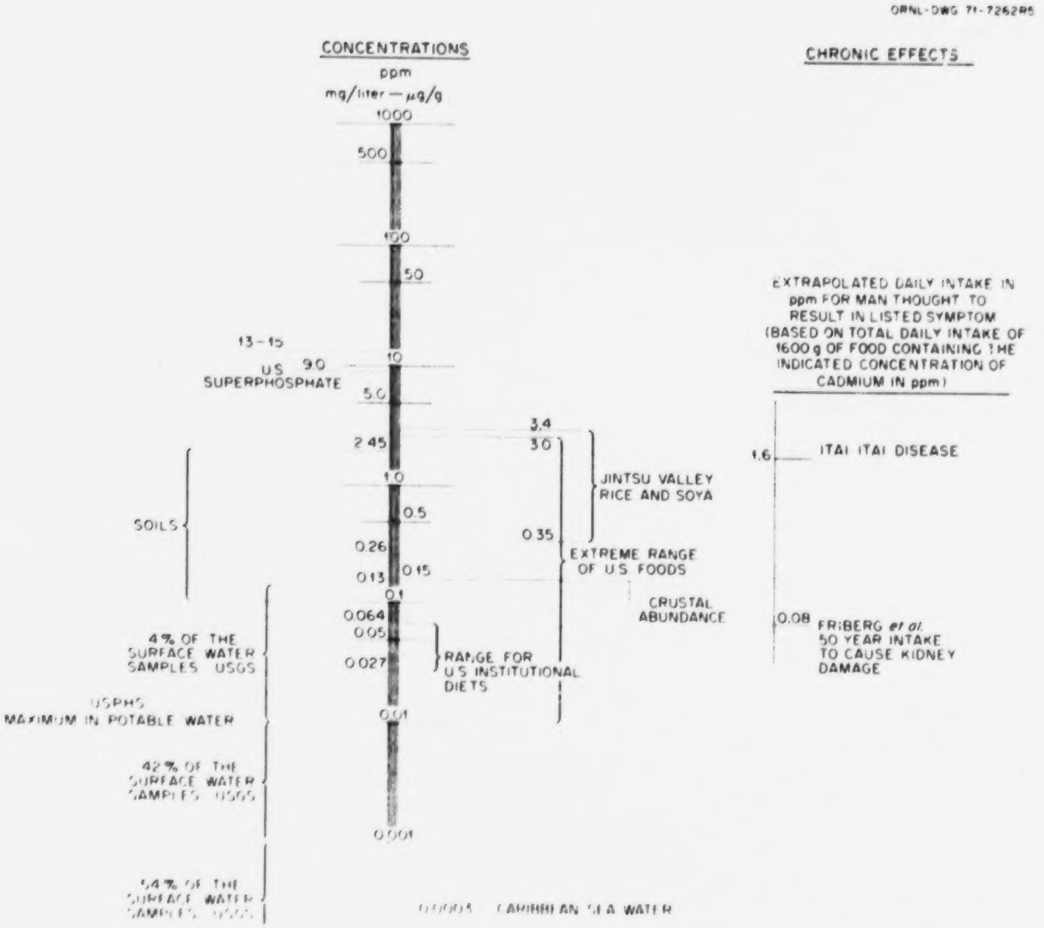


Figure 8.1. Cadmium concentrations of surface waters, soils, and foods and estimated dose levels resulting in various symptoms and effects in humans. Source: Hise and Fulkerson, 1973, Figure VI-1, p. 205.

soils, and foods and gives estimated dose levels resulting in various symptoms and effects in humans. The maximum tolerable content of cadmium in drinking water has been established at 10 ppb by the World Health Organization and the U.S. Public Health Service, but no maximum limits have been agreed on for ambient air or foods (Hise and Fulkerson, 1973).

Apparently, the cadmium content of food is the major source of cadmium for most people. Because the foods ingested are diverse, examination of cadmium movement in a variety of food chains is necessary. The content of a particular food may vary because of (1) differences in the soil in which the food was grown and in the cadmium content of the water supply; (2) the type and amount of fertilizer used; (3) the ability of the plant to absorb, retain, and concentrate cadmium; and (4) the food processing procedures (Hise and Fulkerson, 1973).

The cadmium content of several foods as well as the zinc-to-cadmium ratio is given in Table 8.1. Differences in cadmium content of foods due to regional influences are shown in Figure 8.2. Food plants from the vicinity of a smelter in Annaka City, Japan, had much higher cadmium contents than plants grown in uncontaminated areas (Kobayashi, 1972). Zook, Greene, and Morris (1970) found significant differences in the cadmium content of wheat grains, but they concluded that "no geographical population of the United States should be expected to differ markedly from another in its status of mineral nutrition, solely on the basis of consumption of wheat products." Lagerwerff (cited in Sanjour, 1974) suggested that the principal source of cadmium in vegetables is the natural cadmium content of the soil. Sanjour (1974) supported this contention with the following statements:

1. Air levels of cadmium in rural areas are often unmeasurable.

2. Even in urban areas not near smelters, the air concentrations are low enough not to significantly affect soil concentrations.

3. There are few serious point sources of contamination, and the radius of effect is about 8 to 24 km.

4. Irrigation water generally has a cadmium concentration too low to affect soil concentrations.

In a recent review of cadmium levels in food, Friberg et al. (1975) cited analyses performed by Kjellström in which the cadmium content of Swedish wheat samples, harvested in one geographical area, increased over the years 1916 to 1972. Friberg et al. (1975) suggested that some meteorological, agricultural, or pollution factor, or a combination of such factors, could explain the observed differences in cadmium concentrations. Kjellström et al. (1975) concluded that the increase in the average cadmium concentration in fall wheat may be caused by accumulated effects of air pollution and fertilizers. Statistical analyses performed by Kjellström et al. (1975) did not reveal any correlation between cadmium concentration and yield, average summer temperature, average summer rain, or type and amount of fertilizer applied.

A Swedish study on the contribution of land application of sewage sludge to crop plants suggested that an increased use of sewage sludge will increase the cadmium content of wheat (Linnman et al., 1973). These authors estimated that a moderate increase in the cadmium content of wheat flour in the range of 0.02 to 0.10 ppm would raise average intake of cadmium from cereal to 17 μg per day (assuming an average individual cereal consumption of 166 g/day). However, since for various broad food groups ninth decile figures can exceed the mean consumption by a factor of 3.5, the cadmium intake from cereals may be as high as 58 μg/day for some people.

TABLE 8.1. CADMIUM CONTENT AND ZINC TO CADMIUM RATIO IN SOME FOODS

Food	Cadmium content (ppm wet wt)				Zn/Cd ratio	
	Investigator No. 1[a]	Investigator No. 2[b]	Investigator No. 3[c]	Investigator No. 4[d]	Investigator No. 1[a]	Investigator No. 3[c]
Vegetables						
Potatoes	0.03	0.017	0.038		290	85
Onions	0.01	0.0077	0.012		89	106
Carrots	0.30	0	0.041		17	67
Cabbage	0.07	0	0.009	0.034	26	204
Kohlrabi	0.09			0.052	25	
Beans, kidney	0.07		0.019		12	1120
Beans, string, fresh	0.01				125	
Tomato, fresh	0.03	0.013	0.032		20	45
Tomato, canned	0.02	0.044			6	
Greens	0.06				7	
Peas, green, fresh	0.04				200	
Spinach, fresh	0.45	0.064	0.128		5	71
Beans, string, canned	0.03	0.024			3	
Grains						
Corn, cornmeal	0.12	0		0.033	30	
Wheat	0.15		0.025		430	230
Bread, white	0.22	0.042	0.046		5.5	130
Bread, dark	0.15	0.054			35	
Fruits						
Tangerine orange	0.02		0.003		72	250
Apricot, canned	0.01				5	
Banana	0.03		0.011		7	150
Grapes		0.007				
Apples		0.0065	0.004			52
Meats						
Pork	0.25	0.034	0.016		45	1080
Beef	0.89	0.060	0.054		63	1220
Chicken	1.25		0.027		60	600
Whale			0.33			31
Kidney	0.52	0.43			40	
Dairy						
Milk, whole	0.12	0				
Cheese		0.039				
Butter	0.56					
Eggs, whole	0.09	0.021	0.015			950

(continued)

TABLE 8.1 (continued)

Food	Cadmium content (ppm wet wt)				Zn/Cd ratio	
	Investigator No. 1[a]	Investigator No. 2[b]	Investigator No. 3[c]	Investigator No. 4[d]	Investigator No. 1[a]	Investigator No. 3[c]
Seafood						
Oysters, fresh	3.66		0.62		350	165
Clams, fresh	0.31		0.28		60	67
Shrimp	0.24		0.032		62	220
Tuna	0.21		0.066		87	77
Sardines	0.13	0.066	0.011		150	770
Salt herring		0				
Mackerel			0.018			530
Freshwater fish						
Carp			0.003	0.036		5800
Bass				0.037		

[a] Schroeder et al., 1967.
[b] Rautu and Sporn, 1970.
[c] Ishizaki, Fukushima, and Sakamoto, 1970.
[d] Oak Ridge National Laboratory (unpublished results of analysis of samples contributed by Purdue University).

Source: Adapted from Hise and Fulkerson, Table VI-14, p. 227. Data collected from several sources.

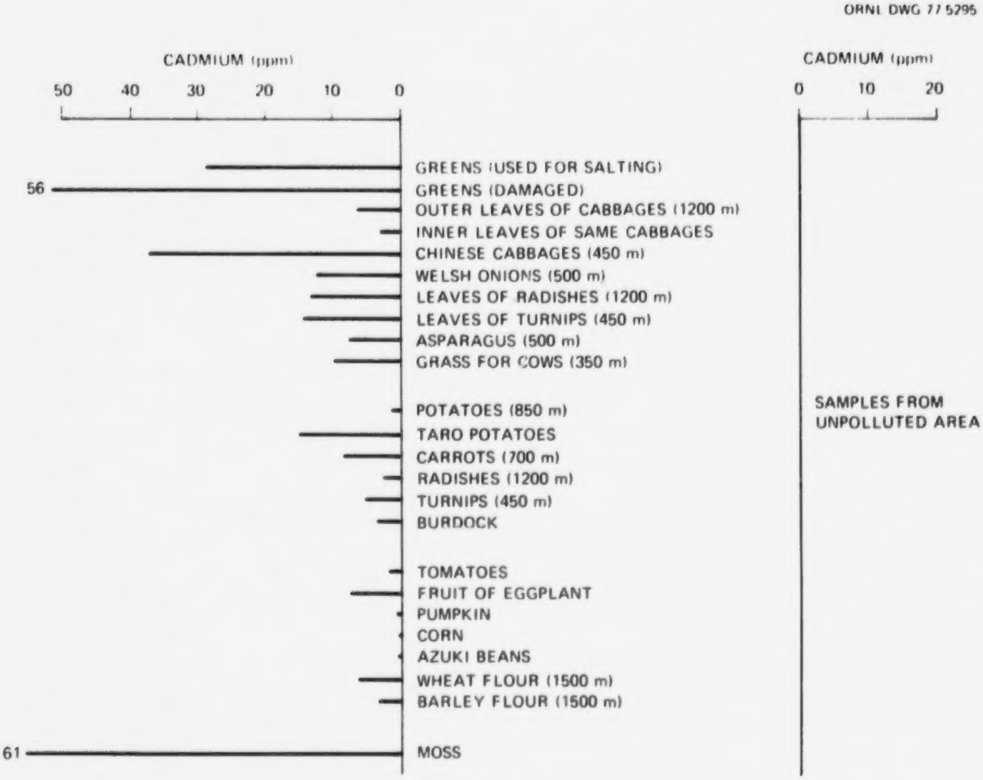


Figure 8.2. Cadmium content in various food samples collected in October 1968 and June 1969 in Annaka City, Japan, compared with unpolluted samples. Numbers in parentheses indicate distances from the smelter. Source: Adapted from Kobayashi, 1972, Figure 5, p. 123. Reprinted by permission of the publisher.

The use of cadmium-containing fungicides has reportedly led to increased levels in plants. After a single application of a cadmium-containing fungicide to apple trees, cadmium accumulated in fruits and decreased in foliage during the growing season (Ross and Stewart, 1969). Although several fungicides contain cadmium, information is lacking on the uptake of cadmium from these fungicides and on its persistence in plants.

In order to understand and evaluate the effects of cadmium, a description of a food web for man is necessary. To identify stages in which bioconcentration, bioaccumulation, and/or biomagnification of cadmium occur, consistent definitions of these terms are necessary (Kneip and Lauer, 1973):

1. Bioconcentration — the ability of the organism to concentrate a substance from an aquatic system
2. Bioaccumulation — the ability of an organism not only to concentrate but to continue to concentrate throughout its metabolic lifetime

3. Biomagnification — refers to the substance being found at successively higher concentrations with increasing trophic levels in the food chain

A simplified food web for man is diagrammed in Figure 8.3. Because man is basically an omnivore, the cadmium content and concentration factors for a number of specific food chains should be estimated. For food web considerations, all ecosystems can arbitrarily be divided into terrestrial and aquatic ecosystems. Data on cadmium movement through these ecosystems are presented in Sections 8.3.3 and 8.3.4.

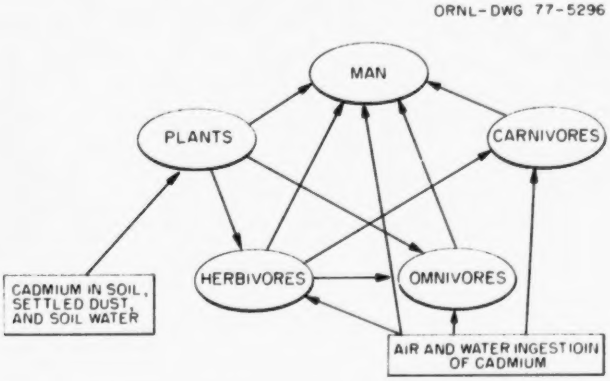

ORNL-DWG 77-5296
MAN
PLANTS
CARNIVORES
HERBIVORES
OMNIVORES
CADMIUM IN SOIL, SETTLED DUST, AND SOIL WATER
AIR AND WATER INGESTIOIN OF CADMIUM

Figure 8.3. Generalized food web for man showing cadmium introduction and movement.

8.3.2 Cadmium in Cigarettes

Because smoking contributes to the body burden of cadmium (Section 6.2.1.1), it is of interest to know whether there have been changes in the cadmium content of cigarettes with time (Friberg et al., 1975). Such a study has been conducted by Linnman, Lind, and Kjellström (1974, cited in Friberg et al., 1975). Eighteen brands of cigarettes produced in the years 1918 to 1968 were analyzed and, with one exception, the concentrations were similar to those found today, with no tendency toward an increase (Figure 8.4).

Friberg et al. (1975) also referred to the work of Elia, Menden, and Petering (1973) in which lettuce leaf cigarettes were examined. The average cadmium content was 1.39 μg per cigarette. These cigarettes were smoked in a smoking apparatus using a standardized procedure. In the mainstream smoke, only 0.01 to 0.02 μg was found — less than is found in ordinary cigarettes. On the other hand, these cigarettes emitted more cadmium via the sidestream than standard cigarettes. In 1972, Menden et al. reported that 38% to 50% of the cadmium from the smoked portion of ordinary cigarettes is present in the sidestream. Thus, cadmium in the sidestream smoke of both lettuce leaf and ordinary cigarettes may present a health hazard to nonsmokers in the vicinity of the smoker, as well as to the smoker (Menden et al., 1972).

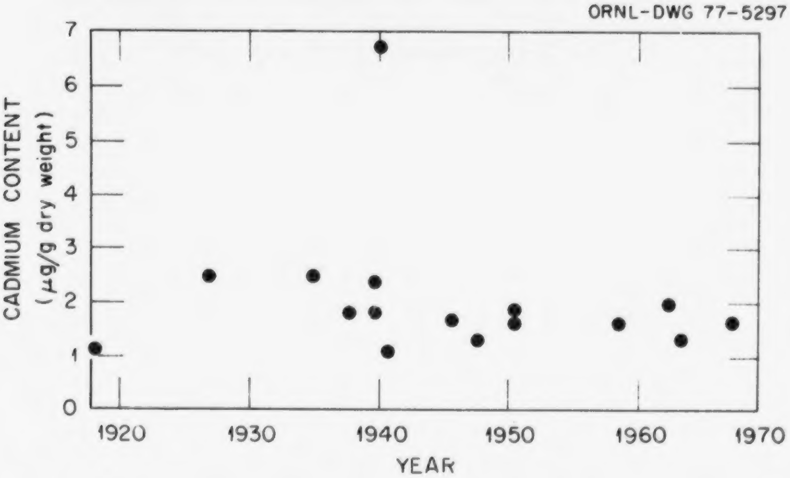


Figure 8.4. Cadmium concentration in cigarettes of different ages. Source: Adapted from Linnman, Lind, and Kjellström, 1974, as cited in Friberg et al., 1975, Figure 3:2, p. 3-24. Reprinted by permission of the publisher.

8.3.3 Terrestrial Ecosystems

Numerous studies have shown that plants take up cadmium; generally, the concentration in plants grown in high cadmium soils is greater than in those grown in low cadmium soils. The cadmium content differs in various parts of the plant; roots often have the highest concentration (Section 4.2.2).

Minerals taken up by plants are distributed throughout the plant and can be returned to the ground either through leaching of the foliage or through removal and decay of portions of the plant. Witherspoon (as cited in Huckabee and Blaylock, 1973) injected ^{109}Cd into tree trunks and two months later found 36.8% of the activity in the branches, 34.6% in the trunk, and 28.5% in the foliage; 0.1% of the activity was leached from the tree by rain. Of this leached activity, 2.7% was in understory vegetation, 68% was in the litter, and 29.3% was in the soil. A study of cadmium cycling in an old field ecosystem ($^{109}CdCl_2$ added in water spray) showed that the initial accumulation was greatest in the soil and litter and that subsequent loss from the litter resulted from decay. Most of the soil radiocadmium was in the surface layers and increased with time due to leaching from the vegetation (Matti, Witherspoon, and Blaylock, 1975).

Thus, cadmium can be leached from vegetation into the soil and litter layers where it is available for uptake by various plants, microbes, and soil invertebrates. In addition, biological breakdown of plant materials introduces cadmium into various decomposer organisms. These soil-dwelling organisms are components of many complicated food chains and the cadmium they take up is potentially available for organisms at the next trophic level. For example, data from an invertebrate grassland food chain (Table 8.2) show that although both an omnivore and a predator contained cadmium, no biomagnification or concentration occurred (Anderson et al., 1974).

Earthworms may represent an important source of cadmium in terrestrial food chains. Cadmium levels in six soils were between 0.23 and 0.80 ppm, whereas in earthworms the concentration ranged from 2.1 to 9.3 ppm — a concentration factor of over 10. This reflects bioconcentration of cadmium in the earthworm; the latter, in turn, may serve as source of cadmium for higher trophic levels (Van Hook et al., 1974). Soil samples consisted of only the top 10 cm of soil with vegetation removed. Cadmium is generally bound to organic matter in the soil and earthworms digest this material; therefore, it is possible that the concentration factors presented in this study may be too high to the extent that reported soil values are too low. In addition to accumulating cadmium, earthworms are responsible for considerable mechanical mixing of soil and thus tend to bring about a uniform cadmium distribution (Anderson et al., 1974).

Because the amount of cadmium taken up by plants depends on soil concentrations, plants growing in the vicinity of smelters often have higher cadmium concentrations. Animals feeding in these vicinities are therefore likely to contain higher than normal concentrations of cadmium. For instance, in the smelter region of East Helena, Montana, mammalian hair

TABLE 8.2. CONCENTRATIONS OF CADMIUM ADDED TO FIELD PLOTS AS $^{109}Cd(NO_3)_2$ IN FOUR GRASSLAND COMMUNITY FOOD CHAIN COMPONENTS

Trophic level	Cadmium concentration (ppm)	Concentration ratio[a]
Predator — *Lycosa* (spider)	0.030	0.71
Omnivore — *Pteronemobius* (cricket)	0.042	0.60
Living and dead vegetation (mean)	0.069	
Living vegetation	0.004	1.0
Dead vegetation	0.135	
Soil	0.004	

[a] Ratio of cadmium (ppm) in trophic level (n) to that in trophic level ($n - 1$).

Source: Adapted from Anderson et al., 1974, Table 5.2.17, p. 124.

contained high concentrations of arsenic, cadmium, and lead; livestock, especially horses, exhibited symptoms of heavy-metal poisoning and contained high levels of cadmium in liver and kidney (Smith and Huckabee, 1973).

A study of cadmium, copper, lead, and zinc contamination at a test farm in the lead-producing region of southeast Missouri showed that the high lead and cadmium content in dust and air was due to smelter stack emissions, ore concentrate spillage, and dust from stockpiled ore. Concentrations in roots were significantly increased at the test farm as were the contents in the washed tops of the plants. Cattle hair, liver, and kidney contained higher cadmium and lead concentrations at the test farm (University of Missouri, 1973). Herbivores may obtain cadmium not only from plant materials, but also, especially in the case of sheep, from direct ingestion of soil.

In a study of a Montana grassland ecosystem near a smelter that processed zinc ore, Munshower (1972) presented data for cadmium concentration in autotrophs and herbivores. Total soil cadmium concentrations were high near the smelter; they decreased with depth in the soil. At soil pH 5.8 more cadmium could be leached (0.87% total) by simulated rain than at pH of 6.4 or 7.2 (0.01% to 0.04%). The cadmium content of grass (1 to 2.4 ppm) was similar in a region near the smelter (1.6 to 6.4 km) and in one farther removed (19 to 24 km). Shrubs and willows contained higher concentrations (5.6 and 13.7 ppm respectively) at the closer sites than at the more distant sites (1.9 and 1.7 ppm). Cadmium was not accumulated to concentrations greater than that of 1 *N* HCl extracts of the soil (50% to 75% to total soil cadmium concentration). Grasshoppers (herbivores) accumulated cadmium to levels above that of the plants on which they grazed; thus, grasshoppers near the smelter contained more cadmium than those farther away. Examination of three species of grasshoppers and their predominant food plants showed cadmium accumulation ratios to be less than 2. For other herbivores examined, "maximum concentrations recorded in cattle, swine, and ground squirrel kidneys were evidence of the accumulation of cadmium in this organ to levels two or more times the cadmium levels found in the food supply of the animal."

Few other studies were found concerning cadmium movement in ecosystems with higher herbivores and omnivores. Lichens in Kokkola, Finland, contained 1 ppm cadmium near a zinc refinery and between 0.3 and 0.4 ppm in other areas (Jaakkola, Takahashi, and Miettinen, 1973). Elk and reindeer also contained measurable amounts of cadmium in the organs examined, although no comparisons were made with animals from other areas. Andren et al. (1973) reported cadmium concentrations in a variety of organisms in different trophic levels of a forest ecosystem (Figure 8.5). Except for insects and earthworms, no group of organisms showed a clear concentration increase in the food chain.

Because grains, seeds, earthworms, and other invertebrates all contain cadmium, a variety of birds are exposed to a range of cadmium concentrations. The cadmium content of starlings (*Sturnus vulgaris*) collected throughout the United States ranged from levels of less than 0.05 ppm to 0.24 ppm (wet weight); 72% of the samples contained the lower limit or less. No comparative data are available, so trends in time and space cannot be established

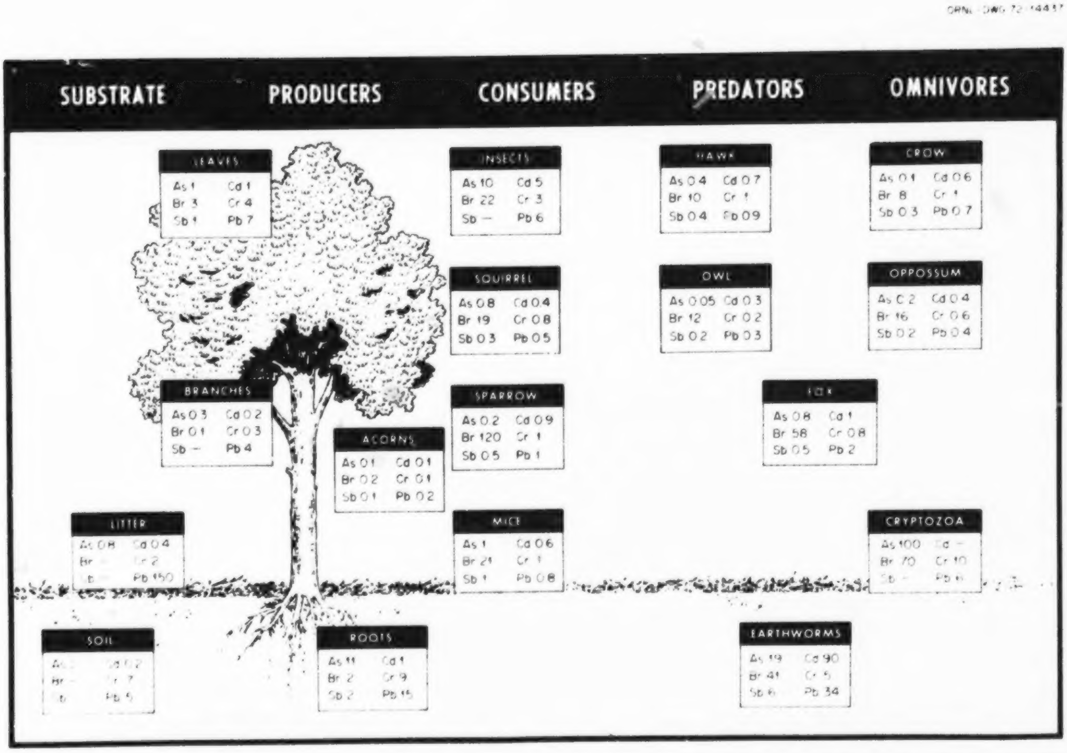

ORNL-DWG 72-14437
SUBSTRATE
PRODUCERS
CONSUMERS
PREDATORS
OMNIVORES
LEAVES
As 1 Cd 1
Br 3 Cr 4
Sb 1 Pb 7
INSECTS
As 10 Cd 5
Br 22 Cr 3
Sb — Pb 6
HAWK
As 0.4 Cd 0.7
Br 10 Cr 1
Sb 0.4 Pb 0.9
CROW
As 0.1 Cd 0.6
Br 8 Cr 1
Sb 0.3 Pb 0.7
SQUIRREL
As 0.8 Cd 0.4
Br 19 Cr 0.8
Sb 0.3 Pb 0.5
OWL
As 0.05 Cd 0.3
Br 12 Cr 0.2
Sb 0.2 Pb 0.3
OPPOSSUM
As 0.2 Cd 0.4
Br 16 Cr 0.6
Sb 0.2 Pb 0.4
BRANCHES
As 0.3 Cd 0.2
Br 0.1 Cr 0.3
Sb — Pb 4
SPARROW
As 0.2 Cd 0.9
Br 120 Cr 1
Sb 0.5 Pb 1
FOX
As 0.8 Cd 1
Br 58 Cr 0.8
Sb 0.5 Pb 2
ACORNS
As 0.1 Cd 0.1
Br 0.2 Cr 0.1
Sb 0.1 Pb 0.2
LITTER
As 0.8 Cd 0.4
Br — Cr 2
Sb — Pb 150
MICE
As 1 Cd 0.6
Br 21 Cr 1
Sb 1 Pb 0.8
CRYPTOZOA
As 100 Cd —
Br 70 Cr 10
Sb — Pb 6
SOIL
As Cd 0.2
Br — Cr 7
Sb Pb 5
ROOTS
As 11 Cd 1
Br 2 Cr 9
Sb 2 Pb 15
EARTHWORMS
As 19 Cd 90
Br 41 Cr 5
Sb 6 Pb 34

Figure 8.5. Metal cadmium concentrations (ppm dry weight) in selected trophic levels of a deciduous forest ecosystem in East Tennessee. Source: Andren et al., 1973, Figure 4.2.15, p. 92.

(Martin and Nickerson, 1973). Information on the cadmium content of each food and on the proportion of each food in the diet is necessary before concentration or accumulation can be determined. No data could be found giving this information for bird food chains.

Studies of the cadmium and other heavy-metal contents of the litter layer in the Crooked Creek watershed near the American Metal Climax, Inc. lead smelter suggested that the high concentrations found "may reduce or eliminate certain bacterial, fungal, and arthropod populations thereby reducing decomposition rates" (Andren et al., 1974).

8.3.4 Aquatic Ecosystems

Several studies of cadmium concentrations in aquatic ecosystems have been reported. Before citing selected data, it is important to determine the information required for development of useful models for the environmental cycling of contaminating minerals. Wolfe and Rice (1972) detailed the following major sets of information necessary for estimating mineral reservoirs in estuaries:

1. Relative amounts of different physicochemical forms of an element or radioisotope in natural waters, their relative stabilities, and the ease of interconversion between the various forms
2. The relative biological availabilities of these different physicochemical forms to various types of biota
3. The trophic structure of the entire ecosystem (particularly the role of microorganisms as sources of metallic elements to consumers in detritus-based food chains, as producers of organic-metal complexes, and as remineralizers of metals previously incorporated into plant or animal tissues)
4. Feeding rates and assimilation efficiencies for carbon and metallic elements at each major trophic interaction
5. Biological retention of metallic elements in the major organisms consumed by man
6. The interactions of variable environmental parameters on reservoir size and transfer rates at each step in the overall system

This information is necessary to determine the extent of mineral cycling for any type of ecosystem. Although some data exist for cadmium on items 1 and 2 for several ecosystems, no studies were found in which all points of information were determined for the same ecosystem.

Phytoplankton is an integral part of many aquatic food chains. Samples from Monterey Bay, California, were found to have increased cadmium concentrations after periods of peak productivity (Knauer and Martin, 1973). Unlike copper, lead, manganese, or zinc, sufficient cadmium was apparently taken up by phytoplankton to decrease the concentration of the metal in the water. During periods of upwelling, metal concentrations in the plankton were high and variable. Wissmar (1972) showed that concentrations of cadmium, copper, and zinc could synergistically interact to decrease carbon dioxide fixation. This finding raises the possibility of toxic effects in rivers or lakes containing such metals. Of course, a variety of other factors such as presence of other minerals, pH, oxygen, and tension, must also be considered.

Brooks and Rumsby (1965) found that cadmium was highly enriched in the scallop (by a factor of 2,260,000) and in the oyster (by a factor of 318,000) as compared with seawater. Actual whole-body levels in the soft parts of these shellfish taken from the Tasman Bay, north of Nelson, New Zealand, averaged 249 ppm dry weight in the scallop and 35 ppm dry weight in the oyster. Cadmium was not detected in the mussel (limit of detection 20 ppm). Essentially all cadmium in the scallop was located in the visceral mass, which includes the gut content, whereas in the oyster most cadmium was located in the mantle, heart, kidney, and striated muscle.

Kopfler and Mayer (1973) attempted to relate water concentrations of various metals in Mobile Bay, Alabama, to their concentrations in oysters.

Bay water contained 0.3 to 0.6 ppb cadmium, while cadmium in oysters from different locales ranged from 0.46 to 1.04 ppm wet weight (overall average 0.62 ppm). Although bioconcentration occurred, high values in oysters could not be correlated statistically with high cadmium values in water. Atlantic Coast oysters contained cadmium concentrations about five times as high (average 3.10 ppm) as the oysters in Mobile Bay. The poor correlation between concentration of cadmium in water and its accumulation in oysters may be due to particulate cadmium ingestion by the Atlantic Coast oyster. Bender, Huggett, and Slone (1972) reported that Chesapeake Bay oysters contained from <0.6 ppm to >2.5 ppm cadmium. They speculated that heavy-metal concentrations in sediments and/or salinity of the water may have affected metal uptake by the oysters.

The experimental addition of sewage sludge and urea fertilizers to salt marsh plots on the East Coast did not affect growth of oysters (*Crassostrea virginica*) or clams (*Mercenaria mercenaria*) but did increase the cadmium content of each (Valiela, Banus, and Teal, 1974). In the Bristol Channel, the cadmium content of *Patella* (clam) was related to physical size; the cadmium content of samples was highest near the largest population centers (Nickless, Stenner, and Terrille, 1972).

Uptake of cadmium by the Mediterranean mussel and benthic shrimp was measured with ^{109}Cd (Fowler and Benayoun, 1974). Radiocadmium concentrations were greatest in the viscera and muscle of both organisms; elimination of the cadmium was very slow.

In an artificial marine microcosm, oysters (*Crassostrea*), clams (*Mercenaria*), sand worms, grass shrimp, and filamentous green algae were exposed to flowing water containing 50 ppb cadmium ($CdCl_2$) (Kerfoot, 1973). Results after 67 days of exposure and 14 days of flushing with fresh seawater are shown in Table 8.3. The largest percentage of total cadmium was found in oysters and sediments.

In summary, there is good evidence that oysters concentrate cadmium above ambient levels. This behavior becomes especially important where the background cadmium is raised, usually as a result of human activity.

Several studies have been made on the cadmium content of fish. Lovett et al. (1972) listed the cadmium content of 406 freshwater fish from New York waters (selected examples in Table 8.4); values were usually below 20 ppb, although values higher than 110 ppb were occasionally found. Fish from the Adirondack area often contained greater than 20 ppb cadmium; these higher concentrations may be related to the higher background cadmium level of this area. Trout of various ages from Cayuga Lake had about the same cadmium content; thus, accumulation could not be demonstrated. Windom et al. (1973) reported that inshore and offshore species of chondrichthyes and osteichthyes had similar cadmium contents. The mean cadmium concentration in deep-sea fish off Great Britain was 50 ppb, although values up to 150 ppb were found (Portmann, 1972).

In aquarium tests, the cadmium content of bluegills after a two-month exposure to nonlethal water concentrations of 0.5 ppb and 8.0 ppb cadmium

TABLE 8.3. OBSERVED RETENTION OF CADMIUM IN SEDIMENT, SHELLS, AND ORGANISMS OF A SYNTHETIC MARINE MICROCOSM

Component	Total weight (g)	Cadmium content (μg)		Cadmium accumulation[a] (μg)	Percent of total cadmium
		Original	Final		
Algae	6.3	2.5	265	262	2.6
Shellfish					
Oysters	236	274.2	3616	3342	32.8
Clams	76	7.6	46	38	0.4
Shrimp	40	3.2	340	336	3.2
Sand worms	12	0.7	54	53	0.5
Shells	439	174.0	225	51	0.5
Sand	5997	119.9	6240	6120	60.0

[a] Final content minus original content.

Source: Adapted from Kerfoot, 1973, Table 2, p. 6.

was 4 ppb and 30 ppb (ash samples) respectively. No further increase in concentration was observed over an additional four-month period. Further accumulation after two months of exposure was not demonstrated even at the toxic cadmium concentrations of 80 and 850 ppb in water (fish contents about 0.1 ppm and 1 ppm respectively) (Cearley and Coleman, 1974).

A direct relationship between cadmium pollution and cadmium content of fish is suggested by the studies of Jaakkola et al. (1972). "Unpolluted" seawater contained 0.1 to 0.2 ppb cadmium as compared to 0.5 ppb in polluted areas. Corresponding concentrations for cadmium in the sediments were 3 to 10 ppm and 15 to 130 ppm. Pike muscle from unpolluted areas contained 2 to 3 ppb compared to 4 to 13 ppb in polluted areas. Kidney and liver samples contained about 10 to 50 times these levels. The trend appears to be toward increased cadmium levels in fish from polluted areas.

In pilot studies, Landner and Jernelöv (1969) could not demonstrate increased cadmium content in guppies over a five-week period in which their only food source was *Tubifex oligochaetes* containing 0, 10, or 20 ppm cadmium. Guppies in water with 100 ppb cadmium did accumulate cadmium; however, when they were transferred to cadmium-free water, the concentration decreased considerably over a four-week period.

Cadmium was detected by Leatherland and Burton (1974) in a variety of organisms from the "not grossly polluted" Southampton water and The Solent (Great Britain); however, no implications regarding specific food chains were stated (Table 8.5). These workers analyzed the muscle from three

TABLE 8.4. RESIDUES OF TOTAL CADMIUM IN FISH FROM NEW YORK STATE WATERS, 1969

Location	Species	Number of fish	Cadmium residue (ppb fresh wt)	
			Mean	Std error
Blue Mountain Lake	Bullhead catfish	3	27.3	6.1
	Rainbow trout	2	24.0	
	Smallmouth bass	1	22.0	
Butterfield Lake	Bowfin	1	16.0	
	Bullhead catfish	3	18.7	0.3
	Largemouth bass	3	20.0	1.1
	Northern pike	4	44.7	13.6
	Rock bass	2	36.0	
	Smallmouth bass	2	21.5	
	Sucker	1	20.0	
	Walleye pike	3	16.3	2.8
Canadice Lake	Bullhead catfish	1	18.0	
	Lake trout	1	<10	
	Pickerel	2	11.5	
	Rainbow trout	2	15.5	
	Rock bass	1	14.0	
	Smallmouth bass	5	13.0	1.5
Hudson River	Atlantic sturgeon	4	91.5	21.7
	Goldfish	3	142.7	21.5
	Shortnose sturgeon	1		
	Striped bass	6	12.2	1.11
Lake Ontario	Black crappie	1	15.0	
	Carp	1	<10	
	Coho salmon	21	13.5	0.8
	Rock bass	4	17.2	3.3
	Smallmouth bass	10	13.6	1.3
	Splake	1	<10	
	Sucker	2	23.5	
	White bass	1	<10	
	Largemouth bass	1	13.0	
Lake Placid	Brook trout	1	23.0	
	Lake trout	2	28.5	
Skaneateles Lake	Rainbow trout	2	16.0	
Spring Brook	Coho salmon	3	16.3	4.9

(continued)

TABLE 8.4 (continued)

Location	Species	Number of fish	Cadmium residue (ppb fresh wt)	
			Mean	Std error
St. Lawrence River	Brown bullhead	3	23.0	
	Muskie	1	30.0	
	Smallmouth bass	6	24.8	8.7
	Sturgeon	11	26.2	3.4
	Walleye pike	4	14.5	1.3
Susquehanna River	Walleye pike	1	64.0	
	Yellow perch	2	51.0	
Trout Lake	Chain pickerel	3	14.3	1.4
	Smallmouth bass	1	17.0	
Upper Saranac Lake	Largemouth bass	2	14.0	
Utowana Lake	Smallmouth bass	2	12.5	
	Rainbow trout	2	17.0	
Waneta Lake	Muskie	1	14.0	
West Canada Lake	Lake trout	2	30.0	

Source: Adapted from Lovett et al., 1972, Table 2, pp. 1286-1289. Reprinted by permission of the Journal of the Fisheries Research Board of Canada.

species of fish and concluded that no apparent bioconcentration of cadmium occurred in these organisms. The cadmium concentration in the fish muscle was 0.03 ppm.

Mummichogs (*Fundulus heteroclitus*), accumulated ^{115m}Cd from synthetic seawater solutions, "but the rate of the whole body accumulation decreased with increasing concentrations of stable cadmium in the medium." The muscle did not accumulate cadmium, but the viscera, liver, and gills did (Eisler, 1974).

It is difficult to generalize about the cadmium content of fish. Some fish seem to concentrate cadmium, but little information is available on bioaccumulation during the lifetime of the organisms. Concentrations for whole fish may be misleading because some organs concentrate cadmium more than others. In addition, the feeding habits of fish may influence the content of cadmium. For instance, in the Severn Estuary, Great Britain, a direct relationship was found between the cadmium content of fish and the proportion of crustaceans in their diet (Hardisty, Kartar, and Sainsbury, 1974).

TABLE 8.5. CADMIUM IN ORGANISMS FROM WATERS OF THE SOUTHAMPTON AND THE SOLENT AREAS OF ENGLAND

Organism	Location	Cadmium content (ppm dry wt)
Algae, Phaeophyceae		
Laminaria digitata	Lee	0.15
Laminaria saccharina	Lee	0.35
Halidrys siliquosa	Lee	0.43
Porifera		
Halichondria panicea	Lee	0.85
Coelenterata		
Tealia felina	Hamble	0.07
Decapoda		
Palaemon elegans	Hamble	0.31
Gastropoda		
Patella vulgata	Netley	2.7
Littorina littorea	Town Quay	0.94
Crepidula fornicata	Town Quay	1.4
Lamellibranchia		
Mytilus edulis	Town Quay	2.5
Cephalopoda		
Sepia officinalis		
Gills	Solent	0.11
Mantle	Solent	0.03
Tunicata		
Botryllus schlosseri	Town Quay	2.7
Pisces		
Anguilla anguilla, muscle	Marchwood	0.03
Marone labrax, muscle	Gilkicker Point	0.03
Platichthys flesus, muscle	Marchwood	0.03

Source: Adapted from Leatherland and Burton, 1974, Table 2, p. 465. Reprinted by permission of the publisher.

A portion of the cadmium released into terrestrial ecosystems eventually reaches aquatic ecosystems and the effects and extent of this input should be determined. In a microcosm experiment, $^{115m}CdCl_2$ was applied to two terrestrial ecosystems in a simulated rainfall and a total radiocadmium budget was determined 27 days later (Huckabee and Blaylock, 1973). The results (Table 8.6) showed that 3.4% and 4.5% of the cadmium entered the aquatic ecosystem. The fate of radiocadmium in the water was followed by a stream tag experiment which indicated that cadmium enters all components of the aquatic ecosystem (Figure 8.6).

TABLE 8.6. DISTRIBUTION OF 284 μCi $^{115m}CdCl_2$ IN MICROCOSMS 27 DAYS AFTER TAGGING

Component	Microcosm A		Microcosm B	
	^{115m}Cd (μCi)	Activity (%)	^{115m}Cd (μCi)	Activity (%)
Terrestrial		93.8		95.5
Moss	28.90	10.2	23.34	7.9
Higher plants	0.38	0.1	0.78	0.3
Litter	43.40	15.3	30.20	10.6
Soil	193.50	68.1	218.00	76.8
Aquatic		3.4		4.5
Water	0.52	0.2	0.56	0.2
Sediment	8.93	3.1	8.63	3.3
Fish	<0.01	<0.1	<0.09	<0.1
Snails	0.22	0.1	0.14	<0.09
Watercress	0.02	<0.09	0.04	<0.09
Plastic liner	7.89	2.8	3.26	1.1

Source: Adapted from Huckabee and Blaylock, 1973, Table 5, p. 137.

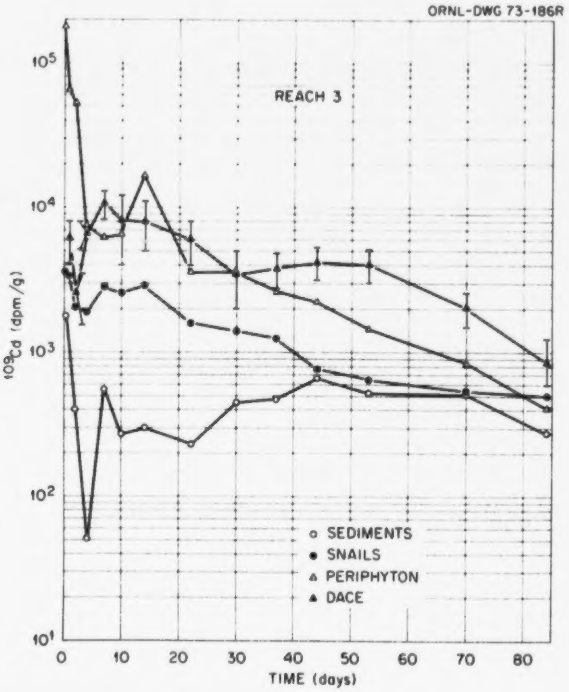


Figure 8.6. Mean activity in stream ecosystem components after labeling with $^{109}CdCl_2$. Composite samples for sediments, snails, and periphyton. Source: Huckabee and Blaylock, 1973, Figure 10, p. 151.

SECTION 8

REFERENCES

1. Anderson, S. H., A. W. Andren, C. F. Baes III, G. J. Dodson, W. F. Harris, G. S. Henderson, D. E. Reichle, J. D. Story, R. I. Van Hook, W. Van Winkle, and A. J. Yates. 1974. Environmental Monitoring of Toxic Materials in Ecosystems. In: Ecology and Analysis of Trace Contaminants. ORNL/NSF/EATC-6, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 95-139.

2. Andren, A. W., B. G. Blaylock, E. A. Bondietti, C. W. Francis, S. G. Hildebrand, J. W. Huckabee, D. R. Jackson, S. E. Lindberg, F. H. Sweeton, R. I. Van Hook, and A. P. Watson. 1974. Ecological Research. In: Ecology and Analysis of Trace Contaminants. ORNL/NSF/EATC-11, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 79-90.

3. Andren, A. W., J.A.C. Fortescue, G. S. Henderson, D. E. Reichle, and R. I. Van Hook. 1973. Environmental Monitoring of Toxic Materials in Ecosystems. In: Ecology and Analysis of Trace Contaminants. ORNL/NSF/EATC-1, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 85-95.

4. Bender, M. E., R. J. Huggett, and H. D. Slone. 1972. Heavy Metals — An Inventory of Existing Conditions. J. Wash. Acad. Sci. 62(2):144-153.

5. Brooks, R. R., and M. G. Rumsby. 1965. The Biogeochemistry of Trace Element Uptake by Some New Zealand Bivalves. Limnol. Oceanogr. 10:521-527.

6. Cearley, J. E., and R. L. Coleman. 1974. Cadmium Toxicity and Bioconcentration in Largemouth Bass and Bluegill. Bull. Environ. Contam. Toxicol. 11(2):146-151.

7. Eisler, R. 1974. Radiocadmium Exchange with Seawater by *Fundulus heteroclitus* (L.) (Pisces: Cyprinodontidae). J. Fish Biol. 6:601-612.

8. Fowler, S. W., and G. Benayoun. 1974. Experimental Studies on Cadmium Flux through Marine Biota. In: Comparative Studies of Food and Environmental Contamination. International Atomic Energy Agency, Vienna. pp. 159-178.

9. Friberg, L., T. Kjellström, G. Nordberg, and M. Piscator. 1975. Cadmium in the Environment — III. EPA-650/2-75-049, U.S. Environmental Protection Agency, Washington, D.C. 218 pp.

10. Friberg, L., M. Piscator, G. F. Nordberg, and T. Kjellström. 1974. Cadmium in the Environment, 2nd ed. CRC Press, Cleveland, Ohio. 248 pp.

11. Hardisty, M. W., S. Kartar, and M. Sainsbury. 1974. Dietary Habits and Heavy Metal Concentrations in Fish from the Severn Estuary and Bristol Channel. Mar. Pollut. Bull. (Great Britain) 5(4):61-63.

12. Hise, E. C., and W. Fulkerson. 1973. Environmental Impact of Cadmium Flow. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 203-322.

13. Huckabee, J. W., and B. G. Blaylock. 1973. Transfer of Mercury and Cadmium from Terrestrial to Aquatic Ecosystems. Adv. Exp. Med. Biol. 40:125-160.

14. Ishizaki, A., M. Fukushima, and M. Sakamoto. 1970. Distribution of Cadmium in Biological Materials: Part 2, Cadmium and Zinc Contents of Foodstuffs. Jpn. J. Hyg. (Japan) 25:207-222.

15. Jaakkola, T., H. Takahashi, and J. K. Miettinen. 1973. Cadmium Content in Sea Water, Bottom Sediment, Fish, Lichen, and Elk in Finland. Environ. Qual. Saf. 2:230-237.

16. Jaakkola, T., H. Takahashi, R. Sininen, K. Rissanen, and J. K. Miettinen. 1972. Cadmium Content of Sea Water, Bottom Sediment and Fish, and Its Elimination Rate in Fish. In: Radiotracer Studies of Chemical Residues in Food and Agriculture. International Atomic Energy Agency, Vienna. pp. 69-75.

17. Kerfoot, W. B. 1973. Cadmium Accrual in a Flowing Marine Microcosm. In: The Use of Flowing Biological Systems in Aquaculture, Sewage Treatment, Pollution Assay, and Food-Chain Studies, J. H. Ryther, ed. Woods Hole Oceanographic Institution, Woods Hole, Mass. pp. 1-10.

18. Kjellström, T., B. Lind, L. Linnman, and C. Elinder. 1975. Variation of Cadmium Concentration in Swedish Wheat and Barley. Arch. Environ. Health 30(7):321-328.

19. Knauer, G. A., and J. H. Martin. 1973. Seasonal Variations of Cadmium, Copper, Manganese, Lead, and Zinc in Water and Phytoplankton in Monterey Bay, California. Limnol. Oceanogr. 18(4):597-604.

20. Kneip, T. J., and G. J. Lauer. 1973. Trace Metal Concentration Factors in Aquatic Ecosystems. Prog. Anal. Chem. 5:43-62.

21. Kobayashi, J. 1972. Air and Water Pollution by Cadmium, Lead and Zinc Attributed to the Largest Zinc Refinery in Japan. Proc. Univ. Mo. Annu. Conf. Trace Subst. Environ. Health 5:117-128.

22. Kopfler, F. C., and J. Mayer. 1973. Concentration of Five Trace Metals in the Waters and Oysters (*Crassostrea virginica*) of Mobile Bay, Alabama. Proc. Nat. Shellfish. Assoc. 63:27-34.

23. Landner, L., and A. Jernelöv. 1969. Cadmium in Aquatic Systems. In: Symposium on Metals and Ecology. Swedish Natural Science Research Council, Stockholm, Sweden. pp. 47-55.

24. Leatherland, T. M., and J. D. Burton. 1974. The Occurrence of Some Trace Metals in Coastal Organisms with Particular Reference to The Solent Region. J. Mar. Biol. Assoc. (Great Britain) 54:457-468.

25. Linmann, L., A. Andersson, K. O. Nilsson, B. Lind, T. Kjellström, and L. Friberg. 1973. Cadmium Uptake by Wheat from Sewage Sludge Used as a Plant Nutrient Source. Arch. Environ. Health 27:45-47.

26. Lovett, R. J., W. H. Gutenmann, I. S. Pakkala, W. D. Youngs, D. J. Lisk, G. E. Burdick, and E. J. Harris. 1972. A Survey of Total Cadmium Content of 406 Fish from 49 New York State Fresh Waters. J. Fish. Res. Board of Can. (Canada) 29:1283-1290.

27. Martin, W. E., and P. R. Nickerson. 1973. Mercury, Lead, Cadmium, and Arsenic Residues in Starlings — 1971. Pestic. Monit. J. 7(1):67-72.

28. Matti, C. S., J. P. Witherspoon, and B. G. Blaylock. 1975. Cycling of Mercury and Cadmium in an Old Field Ecosystem during One Growing Season. ORNL/NSF/EATC-10, Oak Ridge National Laboratory, Oak Ridge, Tenn. 75 pp.

29. Menden, E. E., V. Elia, L. W. Michael, and H. G. Petering. 1972. Distribution of Cadmium and Nickel of Tobacco during Cigarette Smoking. Environ. Sci. Technol. 6(9):830-832.

30. Munshower, F. F. 1972. Cadmium Compartmentation and Cycling in a Grassland Ecosystem in the Deer Lodge Valley, Montana. Ph.D. Dissertation. University of Montana, Missoula, Mont. 106 pp.

31. Nickless, G., R. Stenner, and N. Terrille. 1972. Distribution of Cadmium, Lead and Zinc in the Bristol Channel. Mar. Pollut. Bull. (Great Britain) 3(12):188-190.

32. Portmann, J. E. 1972. The Levels of Certain Metals in Fish from Coastal Waters around England and Wales. Aquaculture (Netherlands) 1:91-96.

33. Rautu, R., and A. Sporn. 1970. Contributions to the Determination of Cadmium Supplied by Foods. Nahrung (East Germany) 14(1):25-31.

34. Ross, R. G., and D.K.R. Stewart. 1969. Cadmium Residues in Apple Fruit and Foliage following a Cover Spray of Cadmium Chloride. Can. J. Plant Sci. (Canada) 49:49-52.

35. Sanjour, W. 1974. Cadmium and Environmental Policy. U.S. Environmental Protection Agency, Washington, D.C. 22 pp.

36. Schroeder, H. A., A. P. Nason, I. H. Tipton, and J. J. Balassa. 1967. Essential Trace Metals in Man: Zinc; Relation to Environmental Cadmium. J. Chronic Dis. (Great Britain) 20:179-210.

37. Smith, R. H., and J. W. Huckabee. 1973. Ecological Studies of the Movement, Fate, and Consequences of Cadmium. In: Cadmium, the Dissipated Element, W. Fulkerson and H. E. Goeller, eds. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. pp. 278-322.

38. University of Missouri. 1973. Study of Lead, Copper, Zinc and Cadmium Contamination of Food Chains of Man. EPA-R3-73-034, Environmental Protection Agency, Durham, N.C. 117 pp.

39. Valiela, I., M. D. Banus, and J. M. Teal. 1974. Response of Salt Marsh Bivalves to Enrichment with Metal-Containing Sewage Sludge and Retention of Lead, Zinc and Cadmium by Marsh Sediments. Environ. Pollut. (Great Britain) 7(2):149-157.

40. Van Hook, R. I., Jr., B. G. Blaylock, E. A. Bondietti, C. W. Francis, J. W. Huckabee, D. E. Reichle, F. H. Sweeton, and J. P. Witherspoon. 1974. Radioisotope Techniques to Evaluate the Environmental Behavior of Cadmium. In: Comparative Studies of Food and Environmental Contamination. International Atomic Energy Agency, Vienna. pp. 23-42.

41. Windom, H., R. Stickney, R. Smith, D. White, and F. Taylor. 1973. Arsenic, Cadmium, Copper, Mercury, and Zinc in Some Species of North Atlantic Finfish. J. Fish. Res. Board Can. (Canada) 30(2):275-279.

42. Wissmar, R. C. 1972. Some Effects of Mine Drainage on Primary Production in Coeur D'Alene River and Lake, Idaho. Ph.D. Dissertation. University of Idaho, Moscow, Idaho. 61 pp.

43. Wolfe, D. A., and T. R. Rice. 1972. Cycling of Elements in Estuaries. Natl. Oceanic Atmos. Adm. (U.S.) Fish. Bull. 70(3):959-972.

44. Zook, E. G., F. E. Greene, and E. R. Morris. 1970. Nutrient Composition of Selected Wheats and Wheat Products: VI. Distribution of Manganese, Copper, Nickel, Zinc, Magnesium, Lead, Tin, Cadmium, and Selenium as Determined by Atomic Absorption Spectroscopy and Colorimetry. Cereal Chem. 47(6):720-738.

SECTION 9

ASSESSMENT OF THE EFFECTS OF CADMIUM IN THE ENVIRONMENT

9.1 INTRODUCTION

Cadmium is a relatively rare element usually present in small amounts in zinc ores. It is commercially obtained as a by-product of the zinc-, copper-, and lead-producing industries. Trace amounts of the metal are also found in fossil fuels, in most soils, and in phosphate rock used for the production of agricultural fertilizers. Cadmium is introduced into the environment chiefly during extraction, refining, and production of metallic cadmium, zinc, lead, and copper; through the wastes generated during other metallurgical processes such as electrolytic plating; through the reprocessing of scrap metal such as cadmium-plated steel; and following disposal by combustion or as solid waste from many consumer goods (paints, nickel-cadmium batteries, plastics, etc.). Cadmium also enters the environment as a result of the agricultural use of cadmium-containing fungicides, phosphate fertilizers, or municipal sewage sludges.

The metal is relatively volatile, and fumes as well as dusts can be a significant source of pollution, especially in industrial surroundings; nevertheless, its major impact on animals and man follows oral ingestion. Tobacco smoke is a significant additional source of cadmium exposure. The potential health risks posed by oral exposure relate to the fact that in spite of the relatively low gastrointestinal absorption of cadmium, its biological half-life is very long. In effect, the metal acts as a cumulative poison, and its long-range chronic effects should be considered. This section assesses the significance of the various modes of entry of cadmium into the environment and the consequent health effects on animals and man. The assessment is based on information collected and discussed in Sections 2 through 8 as well as on some additional references cited at the end of this section.

9.2 NATURE, SOURCES, AND EXTENT OF CADMIUM POLLUTION

9.2.1 Analytical Problems

Analytical methods that require relatively little or no pretreatment of samples and that permit assay of cadmium at levels as low as 1 ng/g (1 ppb), a sensitivity adequate for most purposes, are now available. It must be emphasized that variability of results at such low levels may, to a significant extent, reflect problems of sampling, sample storage, or matrix effects. Both sampling and storage techniques, as well as analytical procedures, therefore require continued careful standardization.

Insufficient attention has been given to the problem of distinguishing between the various chemical forms in which cadmium may occur in the environment or in biological specimens. Occurrence of different forms of cadmium becomes of special importance when, as in many instances, the toxicity or toxicological significance for different compounds may greatly differ. This fact, for instance, makes the choice of a critical total cadmium concentration in the renal cortex a somewhat arbitrary value.

9.2.2 Domestic Production and Import

In 1968 the U.S. consumption of cadmium was approximately 6000 metric tons. About 77% of this total (4600 metric tons) was obtained by refining domestic and imported ores and flue dusts; 9% (540 metric tons) was withdrawn from industrial and government stockpiles; and roughly 14% (860 metric tons) was imported as the metal. Because cadmium is generally obtained as a by-product of zinc-smelting operations, its total production is relatively independent of direct demand; instead, production will vary with the tonnage of zinc ores processed in response to the demand for that metal. To project the entrance of cadmium into the environment over the next 20 or 30 years, one would therefore have to estimate changes in the demand not only for cadmium but also for zinc. Although estimates cannot be made with great accuracy, the use of cadmium is clearly increasing continuously (Section 9.2.3).

9.2.3 Uses of Cadmium

Cadmium is used extensively in a variety of industrial processes. Because little metal is recycled, most of this cadmium ultimately finds its way into the environment.

The rates of consumption in 1968 for most categories of use are expected to double by the year 2000. Some specific processes may consume four times more cadmium by that date (e.g., batteries and electroplating of motor vehicle parts). Projected total U.S. consumption of cadmium in the year 2000 is approximately 14,000 metric tons.

Electroplating thin films on the surface of reactive metals to prevent corrosion is the most important use of cadmium. More than 2700 metric tons of the metal was used in the United States for this purpose in 1968. Other substantial applications include use of cadmium as a pigment in paints, plastics, inks, and phosphors (about 1000 metric tons) and as a stabilizer in plastic goods (about 900 metric tons). United States production of metallurgical alloys, solders, electrical contacts, and solid-state electronic devices required over 200 metric tons of cadmium in 1968. During the same year the manufacture of nickel-cadmium batteries consumed 180 metric tons. Minor amounts of cadmium are also used in photography, rubber processing, nuclear energy, and fungicides. Estimates of these and other miscellaneous uses in the United States in 1968 varied from 285 to 920 metric tons. In 1975 the total U.S. consumption of cadmium was distributed as follows: electroplating, 55%; plastic stabilizers, 21%; pigments, 12%; batteries, 5%; and other uses, 7% (U.S. Environmental Protection Agency, 1976, p. 17).

9.2.4 Flow into the Environment

Contamination of the environment by cadmium-enriched pollutants occurs chiefly during the recovery of cadmium, zinc, lead, and copper from their ores and during the reprocessing of cadmium-plated or galvanized scrap steel. The volatility of the element encourages its loss to the atmosphere during all these operations; fallout and washout return atmospheric cadmium to soils and water. Estimated losses to the atmosphere during 1968 in the United States as a result of primary metal production varied from 100 to 950 metric tons (Sargent and Metz, 1975, p. 140). An even larger quantity of cadmium (about 1000 metric tons) was volatilized that year during reprocessing operations, but only about 10% reached the atmosphere; the balance was collected as dust.

An estimated 100 metric tons of cadmium (calculated as the metal) was released into the environment during 1968 as a result of industrial uses and during consumption and disposal of cadmium-containing products. In addition, between 130 and 1000 metric tons of the metal were contributed by combustion of fossil fuels and from 20 to 200 metric tons by application of phosphate fertilizers. Total losses of cadmium to the U.S. environment in 1968 were estimated at 2000 to 3600 metric tons. Not included in this total is approximately 5000 metric tons of the metal designated for permanent use or considered as waste with unknown disposition.

The association of certain types of cadmium pollution with refining and manufacturing industries results in localization of exposures. In rural and suburban locations distant from point sources the concentration of cadmium in air is normally below the usual level of detection (about 0.001 $\mu g/m^3$). However, near smelting operations average concentrations may range from 0.3 to 3.0 $\mu g/m^3$. Such concentrations decrease sharply with distance from the point source due to dust fall or precipitation. Concentrations of cadmium in urban or industrial air, even in the absence of point sources, are generally greater than those in rural samples because of combustion of fossil fuels; in typical urban neighborhoods airborne particulate cadmium concentrations range from 0.002 to 0.080 $\mu g/m^3$.

Fallout of cadmium dusts near point sources leads to high concentrations of the metal in soil. For example, uncontaminated soil typically contains less than 0.4 $\mu g/g$, but concentrations as much as 100 times higher have been reported from within a 1-km radius of a zinc smelter. In such soils, contaminated mainly by particulate fallout, the vertical concentration gradient of the pollutant is steep, with most cadmium remaining near the surface. Typically, at a depth of 5 to 10 cm, the concentration drops to less than 10% of that at the surface and at 10 to 15 cm to less than 2%.

Soils distant from point sources may be polluted with cadmium from phosphate fertilizers or sewage sludges. The naturally occurring cadmium in phosphate rocks, or that added to sewage, is concentrated during preparation of these materials and some forms may contain from 6 to 75 mg/kg. Such products supply 3 to 45 g of cadmium per hectare when applied at conventional rates. Although uptake of cadmium by plants is controlled by a wide variety of variables such as species, soil pH, and others, the concentration in plants generally reflects the level of the metal in soil. Continued deposition of cadmium, especially on agricultural land, may therefore represent a potentially important problem; this is further considered in Section 9.6.

Cadmium concentration in water varies with season and with distance from polluting sources as well as with the extent of precipitation or adsorption. The removal of cadmium from water into the sediments depends in turn on the nature of the sediments, the presence of particulate matter in suspension, and the chemical composition of the water. Typically, rural waters contain less than 1 μg cadmium per liter, but the level in waters near industrial areas may reach as high as 100 μg/liter. The concentration of cadmium decreases rapidly with distance from polluting source, presumably because of sedimentation. Chemical forms of cadmium in waters are generally not well defined.

To the anthropogenic input of cadmium into the environment must be added input from natural sources such as weathering of rocks and volcanism. These sources may account for an annual input of as much as 5000 metric tons and are presumably responsible for the low background concentrations in air, water, soil, and sediments remote from point sources. The most significant source of cadmium in the environment, however, is modern technological society. The precise amount of cadmium contributed by man is difficult to estimate accurately, but generally about 20% of the amount results from operations in the primary nonferrous metal industry and about 30% from conversion, use, and disposal of cadmium products.

An additional 50% of the input into the environment is derived from activities unrelated to production or use of cadmium metal. Among these, combustion of fossil fuels and use of cadmium-containing fertilizers and sewage sludge deserve special mention. Coal from different sources may contain as much as 2 μg cadmium per gram. In other words, volatilization of the metal contained in 1 metric ton of coal could add 2 g of cadmium to the atmosphere. Even though a major fraction of this metal may be trapped by air pollution controls, this source of environmental contamination is bound to grow with the increased reliance on coal as an energy source. The problem posed by the use of cadmium-containing fertilizers is further considered in Section 9.6.

Automotive transport contributes small amounts of cadmium through the wear of tires; somewhat larger amounts are contributed through the exhaust. Although roadside vegetation may contain increased cadmium concentrations, no strong evidence suggests that the automobile is a major source of cadmium pollution. In general, however, the concentration of cadmium in emissions from anthropogenic sources is appreciably higher than background levels, and the potential for hazardous exposure or adverse ecological consequences is therefore frequently present.

Airborne cadmium constitutes only a small fraction (about 15%) of the anthropogenic cadmium flux. The metal is emitted to the atmosphere primarily as oxide, sulfide, or sulfate particles (U.S. Environmental Protection

Agency, 1975, p. 2-1). Size distribution of the particles varies with source conditions, but typically one-half have diameters less than 2 μm and are therefore readily respirable. The principal sources of airborne emissions are metallurgical processes such as smelting and sintering operations, the reprocessing of galvanized and cadmium-plated scrap, incineration of cadmium-containing plastics, and consumptive uses. Most airborne emissions are deposited on surface soil within 1 km of the point source, but measurable concentration gradients as far away as 100 km have been reported.

Only perhaps 1% of the anthropogenic cadmium flux in the United States originates from point sources as water pollution. The most important water pollution sources are mine and ore beneficiation wastewaters and electroplating wastes. Indirect sources include runoff from land contaminated with cadmium from airborne fallout, fertilizers, and agricultural chemicals. Dissolved cadmium, however, tends to react readily with other constituents of the water, and its concentration is reduced by precipitation and adsorption. Consequently, the metal may accumulate in sediments near its point of entry into the lake or stream. Cadmium adsorbed on fine silts and on suspended matter may ultimately reach marine sediments.

The greatest quantities of cadmium wastes from anthropogenic activities are released into the environment from disposal sites of solid wastes, slimes, and sludges. A substantial fraction of these wastes are residues from air and water pollution control. Only a small percentage of these wastes contains cadmium in concentrations greater than 0.1%. Nevertheless, the usual concentrations in these deposits greatly exceed those of uncontaminated soils so that the deposits represent potentially serious sources of pollution. In the future, economic and regulatory restrictions may induce the recycling of cadmium in many such wastes (Sargent and Metz, 1975, p. 169).

9.3 CADMIUM IN THE HUMAN FOOD CHAIN

Cadmium occurs in most American food plants in concentrations well below 0.1 μg/g; significantly higher values have been found in Japanese rice samples. The exact cadmium level varies with plant species and form and with concentration of the metal in the soil. In normal soils cadmium uptake by plants poses little environmental hazard to man or animals. Crops grown on soils contaminated by smelter emissions or on soils treated with cadmium-rich sewage sludges or cadmium-containing fertilizers and fungicides may contain higher than normal concentrations of cadmium in the edible portions and may thus constitute a direct or indirect source of human exposure. Because use of cadmium is growing and environmental contamination may increase, this problem deserves continued consideration.

Cadmium is present in milk and tissues of domestic animals raised for human consumption, but except possibly for liver and kidney, its concentration falls normally below 0.05 μg/g wet weight and poses little hazard to humans. Some neritic organisms may contain much higher levels of cadmium, depending on proximity to and intensity of sources of pollution. Typically, invertebrate marine animals concentrate the metal from seawater from 1 to 10,000 times. Appreciable consumption of such animals from polluted areas could thus result in significant intake of cadmium. Concentrations in fish are generally lower than in marine invertebrates but may still exceed concentrations in ambient water by a factor of several hundred. In spite of the bioconcentration and bioaccumulation of cadmium in the examples cited, there is no evidence for biomagnification of the metal in terrestrial or marine food chains.

9.4 TOXICITY AND HEALTH EFFECTS OF CADMIUM

This section assesses the toxicity and possible health effects of cadmium in humans. All evidence suggests that the basic mechanisms of toxicity are similar in man and higher animals; therefore, information gained from higher animals may be extrapolated to man.

Acute cadmium poisoning is primarily an occupational problem; the route of exposure is usually via the lungs. Thus, metal workers can receive toxic doses of cadmium fumes during welding operations. Cadmium dusts in the respirable size range also represent a definite hazard. About 25% to 50% of the pulmonary cadmium load may be retained and in acute cases may result in proliferative interstitial pneumonitis, perivascular and peribronchial fibrosis, and possible death from pulmonary edema. The lethal dose of thermally generated fume lies below 3000 mg-min/m^3; arc-generated fume may be twice as toxic. More chronic, low-level exposure to the metal in the atmosphere eventually may also lead to pulmonary pathology (bronchitis and emphysema) as well as to renal damage. Evidence of chronic toxicity has been reported in workers exposed to levels of cadmium significantly below presently accepted standards for the industrial environment (Section 9.5).

Pulmonary exposure of the general population is typically very low except in the case of smokers. The average cigarette may contain 1 to 2 μg cadmium. If about 10% of this cadmium is inhaled and one-half of this inhaled load is retained in the body, a smoker may add as much as 2 μg cadmium to his body burden with each pack of cigarettes. This represents a doubling of the average daily increment of body burden for adults in the United States (Section 9.6). It is worth noting that the side stream of cigarette smoke contains as much cadmium as the inhaled portion.

The major sources of cadmium to which humans are exposed in the normal environment are food and water. In the United States the normal intake from all sources varies from 50 to 75 μg per day (Section 9.6). Fractional absorption from the gastrointestinal tract varies with diet and other ill-defined factors but is typically low (around 5%). Given the fact that the biological half-life of cadmium is very long (tens of years), an average intake of cadmium over a period of 50 years may thus lead to a total body burden of approximately 30 mg. A fraction of 50% or more of this body burden is localized in liver and kidneys, with the renal cortex accounting for perhaps one-third of this fraction. In average American males aged 50, levels as high as 50 μg/g fresh weight have been reported for this tissue. A level of 200 μg/g has been somewhat arbitrarily and provisionally chosen as the concentration at which renal damage may be expected. Many exceptions to this value have been noted; at best, it is only an approximation which is useful until more is known about the renal effects of cadmium. Clearly, even normal chronic exposure to cadmium can lead to an accumulation of the metal in renal cortex; such accumulation represents a significant fraction of suggested critical levels.

Renal cortical levels of 200 µg/g have resulted also from chronic exposure by inhalation to low concentrations of cadmium in air. A variety of renal tubular lesions may result from such concentrations. In extreme cases, such as those associated with itai-itai disease in Japan, where oral intake of cadmium may have reached values as high as 600 µg/day, calcium and vitamin D deficiencies which result in severe bone disease are seen.

Chronic exposure to cadmium does cause lesions in various organ systems, but the renal effects of the metal are probably the most significant ones. No causal relationship has been established between cadmium exposure and human cancer, although a possible link between cadmium and prostate cancer has been indicated. Cadmium injection induces sarcoma in rats but not in mice. Similarly, cadmium has been shown to be teratogenic in rats, hamsters and mice, but no such effects have been proven in humans. Cadmium has also been reported to increase the frequency of chromosomal aberrations in cultured Chinese hamster ovary cells and in human peripheral leucocytes. More recent work indicates no statistically significant difference in the frequency of chromosomal aberrations between controls and individuals exposed to cadmium, including battery workers in Sweden and itai-itai patients in Japan (Friberg et al., 1975). A suggested role of cadmium in human cardiovascular disease remains unproven.

9.5 STANDARDS FOR ENVIRONMENTAL AND OCCUPATIONAL EXPOSURES

Given the appreciable amounts of cadmium introduced into the environment, it is not surprising that significant amounts of the metal are found in the biosphere and ultimately in man. A decision on what constitutes safe levels of cadmium in the environment — levels sufficiently low to provide a significant safety margin against the risk of any normal individual attaining critical levels — is therefore of great importance. This statement of course presupposes agreement on what constitutes a critical level in man. For the present purpose this can best be defined in terms of overt histological and/or functional changes in lungs and kidneys (Section 9.7).

Cases of acute poisoning by inhaled or ingested cadmium or its compounds in the general population usually result only from accidents or suicidal attempts and are relatively rare. Greater danger to the general public lies in long-term low-level exposures leading to appreciable accumulation of cadmium in the body.

As early as 1962 the U.S. Public Health Service sought to minimize such exposure by limiting the concentration of cadmium in approved water supplies to 0.01 mg/liter (U.S. Department of Health, Education, and Welfare, 1962). In 1975, this standard was incorporated into the National Interim Primary Drinking Water Regulations by the U.S. Environmental Protection Agency (*Federal Register*, 1975), making the standard mandatory for all U.S. public potable water systems.

Prior to the 1970s there were no federal standards for cadmium in surface waters other than drinking water. However, the problem was considered at length by the National Technical Advisory Committee in its 1968 report to the Secretary of the Interior (*Water Quality Criteria*, 1968, cited in National Academy of Sciences and National Academy of Engineering, 1973) and by a National Academy of Science–National Academy of Engineering committee on water quality criteria which subsequently revised and augmented the 1968 report (National Academy of Science and National Academy of Engineering, 1973). The latter group concurred with the previously cited public drinking water limit and recommended that, where aquatic organisms and wildlife are involved, cadmium in freshwater streams be limited to 30 µg/liter when the total hardness exceeds 100 mg/liter and to 4 µg/liter for water with a total hardness of 100 mg/liter or below. Upper limits of 50 and 10 µg/liter were suggested for livestock and irrigation uses respectively. To protect marine aquatic life, the concentration of cadmium was restricted to 1% of the 96-hr LC_{50} value for the most sensitive organism present in the water.

No federal standards regulating the cadmium content of industrial water effluents exist, but implementation of such standards by the U.S. Environmental Protection Agency is in progress. Meanwhile, laws in several states restrict the discharge of cadmium in industrial water effluents. The limits in mg/liter are: Illinois, 0.05; Iowa, 0.05; New York, 0.03; Pennsylvania, 0.4; and Wisconsin, less than 1.0. In addition, Missouri, New Jersey, North Dakota, and Oklahoma require effluents to be normal or free of toxic materials (i.e., cadmium must be essentially completely removed). The following states require effluents to meet U.S. Public Health Service standards or to be suitable for various indicated uses: Idaho, Indiana, Kansas, Kentucky, Maryland, Massachusetts, Michigan, Mississippi, Montana, North Carolina, North Dakota, Oregon, South Carolina, West Virginia, and Wyoming (Fulkerson and Goeller, 1973).

At present there are no federal standards for cadmium in ambient air; however, determination of such standards by the U.S. Environmental Protection Agency may be expected in compliance with amended provisions of the Clean Air Act of 1970.

The American Conference of Governmental Industrial Hygienists (ACGIH) recommends threshold limit values (TLVs) for chemical and physical agents in the occupational environment. In 1968 the value for an 8-hr work day was set at 0.2 mg cadmium per cubic meter. Cadmium fume, a more toxic form, was limited to a ceiling value of 0.1 mg/m^3. Subsequently, in 1976, the ACGIH reduced the time-weighted average (TWA) and tentative short-term exposure TLVs for cadmium fume to 0.05 mg/m^3 each and the corresponding values for dust to 0.05 and 0.15 mg/m^3 respectively. A recent document on cadmium (National Institute of Occupational Safety and Health, 1976) proposes a further lowering to 0.04 mg/m^3 determined as the TWA for a 40-hr work week. Chronic toxicity has been observed, however, at even lower exposure levels. In 1971 the Occupational Safety and Health Administration (OSHA) adopted as federal standards for industrial atmospheres values numerically equal to the 1968 ACGIH standards for cadmium fumes and dusts but defined them as 8-hr TWAs and permitted ceiling concentrations of 3 mg/m^3 for dusts and 0.6 mg/m^3 for fumes (*Federal Register*, 1971).

There are no standards for cadmium in foods. It is unlikely, however, that consumption of food containing an average of no more than 0.1 μg/g cadmium presents a significant hazard to humans. In addition, it is the policy of the Food and Drug Administration and the U.S. Department of Agriculture to reject the use of cadmium-containing materials in food preparation and packaging (Fulkerson and Goeller, 1973, p. 381).

9.6 ENVIRONMENTAL HAZARDS POSED BY PRESENT AND PROJECTED CONCENTRATIONS OF CADMIUM IN THE ENVIRONMENT

The total cadmium intake by the average adult human in the United States presently approaches 50 to 75 μg/day, as discussed in Section 9.4. For a typical nonsmoker this uptake is made up of less than 1 μg of airborne cadmium; 2 to 20 μg may be provided by water and about 50 μg by food. Because the efficiency of intestinal absorption is low (Section 9.4), such daily exposure leads to a daily retention of only 1 to 2 μg cadmium. Nevertheless, in the absence of exposure to polluted air or water, and for people in the general population who neither smoke nor are closely exposed to tobacco smoke, food clearly represents the principal source of cadmium. As pointed out in Section 9.4, this daily dose may be doubled by heavy smoking.

The long half-time of cadmium in the body implies that the daily uptake of the metal approximates the net daily increase in body burden. A high proportion of the body burden is concentrated in the renal cortex, for which an approximate critical concentration of 200 μg/g has been suggested (Section 9.4). In order to reach this level it would be necessary to ingest 300 μg cadmium per day for 50 years, according to the calculation of Sargent and Metz (1975, p. 131). According to this criterion, typical intake from unpolluted environments is one-sixth to one-quarter of that required for overt pathological consequences. Thus, no substantial evidence of hazard to the general population from current levels of cadmium in air, water, or food now exists. However, the pronounced tendency of cadmium to accumulate in the body and the relatively small safety margin noted above are adequate reasons for concern over possible future increases in background levels.

Indeed, although concentrations of cadmium in samples of water and air remote from point sources of pollution are low and will probably remain so because of recently introduced pollution control measures, it is likely that concentrations of cadmium in food will increase in the future unless adequate safeguards are taken. Several circumstances favor this development. First, domestic consumption of cadmium is growing at an annual rate of 4% and is expected to more than double by the end of the century (Section 9.2.3). Improved pollution control measures will probably limit further direct contamination of air and water, but increased disposal of cadmium-containing solid waste can result in a general increase of background levels unless adequate storage techniques are employed. Secondly, use of cadmium-containing phosphate fertilizers and sewage sludges is increasing (Sargent and Metz, 1975, p. 8). Application of these materials to crop lands generally increases the uptake of cadmium by the crops and ultimately, therefore, by the human consumer.

There is indeed some suggestion that the cadmium level in certain crops may have risen over the last 50 years. Whether this increase resulted from greater use of cadmium-containing fertilizers or whether it was caused by, for instance, deposition of airborne pollutants originating from industrial sources is not entirely clear. Further uncertainties arise from the fact that cadmium applied in sludge may not remain in soil in forms readily available to plants. In addition, there are ways of reducing the availability of cadmium by, for example, raising soil pH (application of lime). More work is clearly required before the risks of applying cadmium-containing fertilizers can be fully assessed.

Removal of the trace levels (from 2 to 20 µg/g) of cadmium normally present in phosphate fertilizers is technically feasible but not economically practicable. In the case of sludges, quite significant concentrations of cadmium have been found (Sargent and Metz, 1975, p. 11). These cannot always be assigned to specific industrial sources. Nevertheless, it may become advisable to avoid use of sludge as fertilizer except in cases where restrictions on cadmium pollution have maintained metal concentrations at relatively low levels. Ordinances to this effect have been successfully used and may become necessary to forestall leakage of excess cadmium from sewage sludge disposal sites.

9.7 RESEARCH NEEDS

Although the potential significance of cadmium as an environmental toxin is evident, the precise nature and risks of its continued and growing use cannot yet be fully assessed. However, because the metal is a cumulative poison, possible effects of chronic exposure to even low levels in the environment cannot be ignored. Full assessment of these potential risks will require better answers to many questions than we now possess. This section identifies the most important research needs.

9.7.1 Improvements in Identification and Analysis of Low Concentrations of Cadmium

There is a continuing need for standardization and interlaboratory comparisons of rapid and inexpensive methods of analysis of the very low quantities of cadmium encountered in environmental and especially in biological samples. Sample collection and storage are critical factors also requiring standardization. Finally, it must be remembered that in many instances it is not only assay of total cadmium content which may be important, but also identification of the actual cadmium compounds present.

9.7.2 Development of Measures to Minimize and to Adequately Dispose of Wastes

Increased flow of cadmium into the environment may safely be predicted from its growing use as well as from increased reliance on cadmium-containing fossil fuels such as coal. The problem arises not only from direct losses of cadmium during, for example, combustion, but also from the need to dispose of the increased amounts of cadmium-containing wastes. Measures to minimize such wastes should be an integral part of current coal conversion research projects. Moreover, because the cited changes will tend to increase background levels of cadmium and reduce the already relatively small existing margin of safety, it is essential that adequate background monitoring facilities be maintained.

Uncertainties about the increased cadmium levels in crops from lands treated with sewage sludges and other cadmium-containing materials need to be resolved. This problem becomes especially important as difficulties increase in finding an adequate means of disposal for cadmium-rich sludges.

9.7.3 Further Information Related to Biological Effects

In general, in toxicological and metabolic studies with cadmium it is essential to keep in careful focus the extent to which nutrition may alter the effects of the metal. Factors such as the protein content of food or the presence of trace nutrients such as zinc or calcium may alter absorption and/or distribution of cadmium in the body. In addition, however, the possible interaction of cadmium with other toxic substances must also be considered. These include lead and arsenic, which are likely to accompany cadmium in pollution from industrial sources. In the past, most experimental studies with cadmium have dealt with the effects of cadmium alone.

9.7.3.1 Mechanism of Intestinal Absorption — Food and water provide the major portion of the cadmium to which the general population is exposed; fortunately, net fractional absorption to the metal from the gastrointestinal tract only amounts to approximately 5%. This value is influenced by diet and possibly other factors, and more precise information is needed. It is obviously of major significance to the body burden whether fractional absorption amounts to 2% or 10%. Before factors controlling the rate and extent of cadmium absorption can be evaluated, more information is required. Topics of prime concern here are, for instance, the kinetics of cadmium absorption and the mechanism whereby dietary constituents alter this process.

9.7.3.2 Measures of Exposure and Body Burden — Unlike lead, where measurements of delta-aminolevulinic acid excretion in urine or lead in blood can provide an early estimate of exposure, no such measure is readily available for cadmium. The fraction of total body cadmium in blood is small and reflects recent exposure as much as total body burden; the toxicological significance of blood cadmium levels is not clearly established. Usefulness of cadmium levels in hair for monitoring purposes also remains in question. Variations in urinary solute patterns, for example of proteins or amino acids, have been investigated but are not informative in cases of mild or early exposure or have been reported only in isolated cases. Excretion of β-2-microglobulins correlates reasonably well with indices of prolonged exposure (Kjellström, Evrin, and Rahuster, 1977); it provides no direct measure of body burden or of only limited exposure.

Assay of tissue biopsies might provide useful information, but it is obviously inappropriate as a routine procedure. Noninvasive estimation of cadmium levels by activation analysis has been tested in a few instances but requires relatively complex and expensive instrumentation. The same disadvantage applies to the possible use of isotope dilution techniques employing a stable cadmium isotope; an additional problem arises because the tight binding of cadmium by various tissue constituents may cause isotopic equilibration of cadmium in the body to be very slow. In summary, better procedures are clearly required for monitoring body burden of cadmium and for quantitating exposure to the metal before screening procedures for populations at risk can become meaningful.

9.7.3.3 Toxicological Significance of Cadmium Concentrations in Tissues — At present, not only is the mechanism of cadmium toxicity unclear, but the toxicological significance of a given tissue level cannot be properly evaluated. Interaction with other trace metals such as lead and zinc will alter the normal reactions of cadmium in the body; environmental pollution, of course, seldom involves cadmium as the only metal. In addition, cadmium is stored in tissues in a variety of bound forms, such as metallothionein and other metalloproteins, whose precise nature, function, and toxicity are unknown. In the absence of this information, a total tissue concentration like the somewhat arbitrarily suggested critical level of 200 μg cadmium per gram renal cortex can have only limited significance. Even the function of metallothionein in cadmium metabolism, if indeed it has any, remains unclear in spite of the extensive work in that area.

In evaluating the toxicological significance of a given body burden, one must consider not only the general population but also specific subgroups which may be at special risk. Thus, the evidence that cadmium may alter pancreatic function implies that it may pose a special threat to diabetics or pre-diabetics. Similarly, the transplacental movement of cadmium necessitates further study of the possible consequences of fetal accumulation of the metal. In general, genetic, teratogenic, and carcinogenic effects of different cadmium levels need to be further explored (Fleischer et al., 1974, p. 300; Hiatt and Huff, 1975).

SECTION 9

REFERENCES

1. Federal Register. 1971. Occupational Safety and Health Standards. 36:15104.

2. Federal Register. 1975. National Interim Primary Drinking Water Regulations. 40:59566.

3. Fleischer, M., A. F. Sarofim, D. W. Fassett, P. Hammond, H. T. Shacklette, I.C.T. Nisbet, and S. Epstein. 1974. Environmental Impact of Cadmium: A Review by the Panel on Hazardous Trace Substances. Environ. Health Perspect. 7:253-323.

4. Friberg, L., T. Kjellström, G. Nordberg, and M. Piscator. 1975. Cadmium in the Environment — III. EPA-650/2-75-049, U.S. Environmental Protection Agency, Washington, D.C. 218 pp.

5. Fulkerson, W., and H. E. Goeller, eds. 1973. Cadmium, the Dissipated Element. ORNL/NSF/EP-21, Oak Ridge National Laboratory, Oak Ridge, Tenn. 473 pp.

6. Hiatt, V., and J. E. Huff. 1975. The Environmental Impact of Cadmium: An Overview. Intern. J. Environ. Stud. 7:277-285.

7. Kjellström, T., P. E. Evrin, and B. Rahuster. 1977. Dose-Response Analysis of Cadmium-Induced Tubular Proteinuria. Environ. Res. 13:303.

8. National Academy of Sciences and National Academy of Engineering. 1973. Water Quality Criteria 1972. EPA•R3•73•033, U.S. Environmental Protection Agency, Washington, D.C. 594 pp.

9. National Institute of Occupational Safety and Health. 1976. Criteria for a Recommended Standard — Occupational Exposure to Cadmium. HEW Publication No. (NIOSH) 76-192.

10. Sargent, D. H., and J. R. Metz. 1975. Technical and Microeconomic Analysis of Cadmium and Its Compounds. EPA-560/3-75-005, U.S. Environmental Protection Agency, Washington, D.C. 209 pp.

11. U.S. Department of Health, Education, and Welfare. 1962. Public Health Service Drinking Water Standards. Public Health Service Publication No. 956, Washington, D.C. 61 pp.

12. U.S. Environmental Protection Agency. 1975. Scientific and Technical Assessment Report on Cadmium. EPA-600/6-75-003, Washington, D.C.

13. U.S. Environmental Protection Agency. 1976. Summary Characteristics of Selected Chemicals of Near-Term Interest. EPA-560/4-76-004, Washington, D.C.